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2 June, 1911.

A TEXT-BOOK OF MEDICINE

FOR STUDENTS AND PRACTITIONERS

VOLUME I

A TEXT-BOOK OF MEDICINE

FOR STUDENTS AND PRACTITIONERS

BY

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Fourth American Edition

TRANSLATED BY PERMISSION FROM THE SEVENTEENTH REVISED GERMAN EDITION

WITH EDITORIAL NOTES, ADDITIONAL CHAPTERS, AND A SECTION ON MENTAL DISEASES, BY

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WITH SIX PLATES, THREE OF WHICH ARE IN COLOR, AND TWO HUNDRED AND TWENTY-FOUR ILLUSTRATIONS IN THE TEXT

VOLUME I

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PREFACE TO THE AMERICAN EDITION

THE special customs and habits of a people are evidenced even in its medical life. The facts and underlying principles of science, naturally, are common to all nations. In addition, however, medical science and, more particularly, the everyday medical practice of each country manifest also special characteristics. Hence, every physician may learn from the practitioners of other countries something new to broaden and better his own views. This is due not only to the fact that diseases vary in different lands, but also to the further circumstance that, owing to the diversity of climate and other factors, identical diseases present certain distinct variations. The individual national characteristics of the physicians themselves cannot be entirely ignored.

The foregoing considerations have convinced me of the utility of a people supplementing its scientific literature by translations from foreign works, and I have therefore readily given my consent to the publication of an American edition of my "Lehrbuch der speziellen Pathologie und Therapie der inneren Krankheiten."

In both the United States and England there is no lack of good text-books of medicine, but, nevertheless, the American students and physicians may be interested to learn the present German views of the pathology and therapy of medical diseases. My text-book has found wide circulation and recognition in Germany as well as in other countries, and I may therefore assume that it has not altogether failed of its purpose to give a thorough and reliable survey of the present status of internal medicine. I have always been happy to be a clinical teacher, for teaching was ever a pleasure to me and never a task. This accounts, perhaps, for my success in being able clearly and distinctly to express whatever there was to tell about individual diseases. My text-book is concerned with the large and extensive domain of internal medicine. Nowadays no one dares profess to be equally at home in all branches of this study. The experienced reader will observe that some sections, as, for example, Diseases of the Nervous System, have been more fully and more originally presented than have others. Despite this fact, the teacher of internal medicine should, to a certain extent, command a knowledge of all the branches that come within the field of his instruction,

and I therefore hope that my book will be found throughout to present at least the more important facts and views, in conformity with the present status of medical knowledge. The practical end has not been my main purpose in writing. It would be sad, indeed, if the physician should desire to learn and know only that which he could use in his practice. Only the physician who takes a scientific theoretical interest in his work, and who remains conscious of the close relations that always exist between medical science and the important questions of general biology, can find happiness and contentment in his calling. We physicians must never forget that many branches of human physiology can be marked out only with our assistance. Therefore, I have always regarded it as one of the chief duties of a clinical teacher, as well as of a scientific text-book, in addition to the requirements of practice, also to bear in mind the connection between medical science and general biology, and to arouse the interest of the coming physicians in this subject. A scientific pathology is possible only when based upon physiological principles.

I trust that my book, in its new form, will still receive a friendly welcome from its American and English readers. May it, in a small measure, at least, serve to unite two nations in a domain where, after all, there can be nothing that estranges—the promotion of knowledge and social progress.

ADOLF VON STRÜMPELL.

VIENNA.

AUTHOR'S PREFACE TO THE SEVENTEENTH GERMAN EDITION

THE extent to which my text-book has been used, both in Germany and in other countries,[1] justifies me in the assumption that it has exercised some little influence upon the professional thought and practice of numerous medical readers, and imposes upon me the obligation of employing every opportunity for the improvement and perfection of my work. Yet I must confess that this task seems more difficult to me with every new edition of the book, for scientific workers are making deep and extensive investigations into the many subdivisions of internal medicine, with such zeal and skill that it becomes difficult for any one man to keep step with the unceasing progress of investigation in this wide domain, and to maintain that complete and universal command of facts and theories which is essential for a presentation of the subject, which shall be at once brief and true to the present standpoint of science. The author has often been painfully sensible of these limitations of his personal knowledge and ability, but he hopes that this will be regarded not merely as subject for criticism, but also as an excuse and an incentive to clemency, in case a reader who is expert in any specialty finds here and there an omission or an error. Above all, I beg the reader to consider that no text-book such as the present one ought to replace, or could replace, the vast material of the larger reference books. My purpose was not to collect all the facts of pathology which have been discovered up to date, nor all the methods of treatment which may have been recommended, wisely or unwisely, nor all the theories or views which have been propounded. My wish was to give a complete presentation of the essentials of our present knowledge and views with regard to the various diseases, from a scientific and individual standpoint; and I desired particularly to impart to the reader an insight into the origin and relation of the various morbid phenomena. To this end I have brought the facts of clinical experience into the closest possible relation with the data of pathological anatomy and of general pathology, and have endeavored, also, in discussing therapeutics, to deduce from the nature of the symptoms a basis for rational medical opinion and treatment, although I have not undervalued the importance of simple experience. A text-book intended for students and a larger circle of graduate readers ought not to incline too decidedly toward any one of the prevailing currents of scientific opinion. It should, however, acquaint the reader to such an extent with the questions at present under

[1] So far as I have been informed, translations of my text-book have appeared in the following languages: French, English, Italian, Spanish, Russian, modern Greek, Turkish, and Japanese; and some of these translations have had several editions.

discussion relating to internal medicine, as to give him at least a fair start in his further studies.

The completion of this, the seventeenth edition of my text-book, has unfortunately been delayed by my transfer of residence to Vienna. In spite of many demands made on my time by my new activity, I have endeavored, to the best of my ability, to bring the book to the level of contemporary medical knowledge by making numerous changes and additions in all parts of it. I have again laid greatest stress upon the presentation of clinical phenomena as they appear to the physician, separately and in combination, in the course of disease. The detailed description of many important methods of examination must be left to the special text-books. We cannot emphasize and recognize too often how much our diagnostic knowledge has gained in certainty and precision through these methods of examination, and how much we as physicians should strive to give all patients the benefit of this progress. The *only* disadvantage resulting from this is, that the major factor of the examination shifts more and more from the bedside to the laboratory, with a resulting decrease in the practice of and interest in the observation of the purely clinical phenomena. I have repeatedly noted that the younger generation of medical men, however well trained in bacteriological, chemical, and microscopic methods of examination, shows a remarkable insecurity and want of practice in clinical observation and diagnosis. Therefore, I believe that, for scientific as well as for practical reasons, we clinical teachers should not lose sight of the purely clinical phase of instruction, besides the modern achievements in the field of diagnosis and pathological physiology.

The accusation is often made that "what the text-books tell us" does not fully correspond to reality. I trust that my text-book does not deserve this criticism. It is the outgrowth of an unceasing, broad, clinical activity, and is much more a product of the hospital ward than of the study. It would, nevertheless, be unfair to expect that all the unlimited and numerous possibilities and differentiations occurring in the course of diseases should be mentioned. The presentation of a text-book can, after all, in a certain sense only be an abstract with which individual cases are to be compared in order that their peculiarities may be determined. The critical examination of a case, with reference to average medical experience, constitutes one of the chief attractions of clinical observation. However, such comparison is possible only when the essential phases of the common course of diseases are ever present in the physician's mind. It is the aim of this text-book to present these essential phases, and if I have accomplished this, the book will be of help to the physician even after he has recognized that a simple rule rarely embraces all the variations of real facts.

A. Strümpell.

Vienna.

EDITORS' PREFACE

It is just a quarter of a century since the first edition of our translation of Professor Strümpell's Lehrbuch, then in its second edition, was published —a period of time long enough to have seen many text-books appear, flourish, and pass into oblivion—yet each of the fifteen editions of the original which have appeared since that date has shown more freshness and vigor than most of the latest candidates for medical favor. Our study of the successive editions, in the preparation of the various editions of our translation which have been called for, has impressed us more and more with the vast clinical experience, the wide knowledge and sound judgment of the author, his familiarity with the great progress in all branches of internal medicine, his clearness of exposition, and his admirable critical faculty in selecting and emphasizing the facts which he presents to the student in the moderate compass of these volumes. Long familiarity with the work has bred, contrary to the old adage, a greater admiration for it and a deeper conviction that it has, during this time, kept its place as the chief text-book of internal medicine.

Of the present edition we are the editors and not the translators, but an effort has been made, as in the former editions, to keep as close to the original as seemed consistent with clearness. Doses and temperatures have been given in apothecaries' weight and the Fahrenheit scale, corresponding approximately to the metric values of the original. As far as was possible we have tried to follow the United States Pharmacopœia in the prescriptions mentioned, except that we have retained certain familiar names of drugs instead of adopting the innovations of the last revision. We have also substituted specimens of handwriting in English in place of the originals in German script. We have retained a large part of certain chapters and some of the editorial notes contributed to the first edition by Dr. F. C. Shattuck, the former editor; but, in justice to him, it should be understood that he is in no way responsible for any errors or shortcomings in the present edition, although we gladly and gratefully acknowledge our indebtedness to him. We have ourselves added various notes, most of them distinguished by our respective initials. We have also inserted brief chapters on The Fourth Disease, Malta Fever, Rocky Mountain Fever, and Pellagra.

It has not been the custom to include a section on mental diseases in the

ordinary text-book of internal medicine. This exclusion, however, seems to us wholly unjustifiable in view of the great importance of the subject and the necessity for some knowledge of it by the general practitioner. We have therefore added such a section, the aim of which is to give a brief account of the commoner forms of mental disease not already discussed by the author. This account is, of course, not intended to be exhaustive or to consider all the forms of mental disease. For such considerations the student must refer to the special treatises. In this section we have followed in the main the classification of Kraepelin, now so much in vogue in this country, in order to avoid confusing the beginner by any less familiar nomenclature.

H. F. Vickery,
Philip Coombs Knapp.

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A TEXT-BOOK OF MEDICINE

VOLUME I

I. ACUTE GENERAL INFECTIOUS DISEASES

CHAPTER I

TYPHOID FEVER

(Typhus abdominalis. Enteric Fever. Ileotyphus)

Ætiology.—The cause of typhoid fever is an infection of the body by a definite pathogenic bacillus, the "typhoid bacillus" discovered by Eberth and Koch, and later made better known by the researches of Gaffky and others. Their length (see Fig. 1) is about one third the diameter of a red blood globule, and their breadth equals one third their length, but sometimes they may grow out in long fibers. It is not yet certain whether spore formation takes place in their interior or not. The typhoid bacilli show a very active spontaneous movement in water, caused by very fine filiform threads which Löffler was the first to demonstrate on the surfaces and the ends of the rods. The typhoid bacillus is closely related to the Bacterium coli communis (Escherich), but differs from the latter by not fermenting grape sugar and not curdling milk, though acidulating it. Colon bacilli, in contrast to typhoid bacilli, produce indol in meat bouillon, generating a foul odor. The most important differential method depends, however, on the different behavior of the two forms of bacilli toward the blood serum of a typhoid patient (see under serum diagnosis). The special characteristics of the typhoid bacillus, in its growth on various nutritive media, cannot be here detailed. Attention is, however, called to the fact that typhoid bacilli can also thrive if deprived of oxygen, which fact enables us to understand their increase within the intestine.

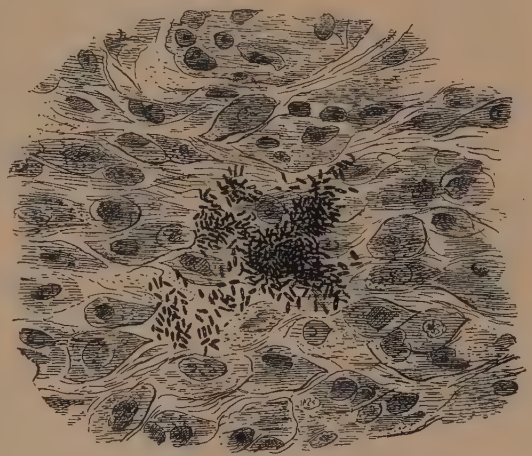

FIG. 1.—Typhoid bacilli. Section from the spleen. 800 : 1. (From FLÜGGE.)

The typhoid bacilli have thus far been found chiefly in the typhoidal infiltrations in the intestine, where they lie between the cells, and also in single foci in the mesenteric glands, the spleen, the liver, the kidneys, the pleura, the

meninges, in typhoidal foci in the bones, in muscle abscesses, in abscesses of the thyroid gland, in the inflamed and enlarged testicle, in the gall bladder (where the bacilli may remain alive for years without producing symptoms), etc. They are often found in the stools of typhoid patients and also in the fluid obtained by puncture of the fresh splenic tumor (in one quarter to one third of all cases), in the gall bladder, sometimes in the urine, also in the blood taken from a spot of roseola, and finally—a fact of great practical importance—they are present in the blood of typhoid patients.

Numerous attempts have been made to produce typhoid fever artificially by introducing pure cultures of the typhoid bacilli into the bodies of animals, but the results of these efforts have not yet proved perfectly harmonious. The main cause of the discrepancy is probably that animals are in general very slightly susceptible to the disease. At any rate, the attempts at artificial infection up to this date have proved successful only in cases when the animals subjected to the experiment (rabbits, guinea pigs) have received large amounts of the typhoid bacilli directly into a vein or into the abdominal cavity (E. Fränkel and Simmonds), or when the bacilli have been introduced directly into the duodenum (A. Fränkel). Even here we have to do rather with the intoxication of the animals than with an actual infection. For the pathological changes of typhoid fever are but little developed in the animals, and the injected bacilli themselves appear to be for the most part destroyed within the body of the animal experimented upon (Flügge and Sirotinin, and others). Normal body fluids have the ability to destroy a certain number of the typhoid bacilli that have entered the organism ("bactericidal power"), and to dissolve them. This solution of the bacteria is the main source of the poisons that lead to the intoxication in typhoid fever. Attempts to produce the disease by mixing the dejecta of typhoid patients with animals' food have thus far been invariably unsuccessful.

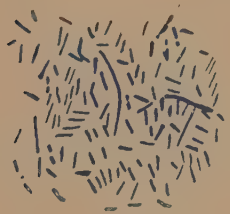

Fig. 2.—Typhoid bacilli. Pure culture.

Investigation of the ætiology of typhoid fever must be directed to ascertaining in what manner and through what channels the specific typhoid bacilli penetrate into the human body, and what circumstances are then essential to their further development and to the display of their pathogenic properties.

It is almost universally believed that typhoid bacilli, as a rule, do not have any permanent existence outside the human body. Sometimes, under favorable conditions, they may remain alive for months in a dry state (as in linens or clothes) as well as in moist earth, stagnant water, etc. Often the conditions essential to an abundant development and transmission of the bacilli arise in certain places, and thus make it possible for a greater or less number of persons to absorb the pathogenic poison, and, as a result, to be attacked by typhoid fever. In this way occur the numerous greater or smaller epidemics of typhoid fever in contrast to the sporadic cases, which are likewise possible, and are not infrequent. If an epidemic of typhoid appears in a place till then entirely free from the disease, we must always refer it to an importation of the disease germs, and seek their source in some previous case of typhoid. In some manner, therefore, the typhoid poison must come from patients already infected with the disease. The stools of the typhoid patients are undoubtedly

the greatest factor in this connection, but the urine also often contains a great number of virulent typhoid bacilli. Of especial importance is the fact that, in a typhoid epidemic, there are numerous ambulant individuals who carry and discharge typhoid bacilli—individuals who do not seem to be seriously ill and complain of only indefinite symptoms, or are apparently entirely well. It is obvious that such "bacillus carriers" are often greater factors in the spread of the disease than the really sick patients who are in bed and isolated. It is also of epidemiological significance that individuals who have recovered from typhoid fever may for months [and even years] discharge virulent typhoid bacilli in their dejecta.

As to the exact manner of transmission of the disease (i. e., the way in which the typhoid bacillus enters the body of a well person from the external world) there were, till recently, widely differing views. There were chiefly two contrasting theories, called, respectively, the "ground-soil" and the "drinking-water"[1] theories. According to the former, which was maintained principally by Pettenkofer and his pupils, the ground soil was regarded as the chief place of development for the schizomycetic fungus of typhoid fever. The poison in the stools must first be changed by the soil before it becomes infectious. The "ground air," which is continually rising, carries the poison. The chief support of the ground-soil theory, beyond the results of comparing the character of the soil with the extent of the epidemics, consisted in the proof which Buhl and Pettenkofer have given (taking Munich as an example, and later Berlin and other places) that a relation exists between the variations of the standing water in the soil and the frequency of typhoid cases. It appears that, when the water stands high (near the surface), fewer cases occur, and when it falls below the mean height cases are more numerous. Pettenkofer explained this relation by the fact that the level of the ground water is certainly an index of the moisture and other conditions of the soil upon which the development of the typhoid bacilli depends.

This "ground-soil" theory is no longer tenable. The observations of Pettenkofer only indicate that the upper strata of the soil are but partial factors in the spread of the typhoid bacilli. Above all, it is highly improbable that the typhoid poison enters the organism by inhalation. All experiences show that the typhoid bacilli commonly gain entrance by being swallowed; it is unquestionable that the most common source of infection is water used for drinking and domestic purposes. This may become infected with typhoid bacilli in the most varied ways. In numerous typhoid epidemics of recent years, a definite relationship between the spread of the disease and the source of the water has been demonstrated. In addition, the typhoid bacilli have often been found in the suspected water. The infection depends, in many cases, on well water that becomes tainted from near-by privies or from other sources. It may also come from running water. Thus, for example, we so strikingly often have outbreaks of typhoidal disease among the employees in the Oder River service at Breslau. Of especial importance, however, are those epidemics of typhoid which arise from infected water flowing through pipe-mains into towns—the sources of the water having been polluted and the filtra-

[1] Compare with what follows the statements concerning the ætiology of cholera, in which disease the same disputed points were in the past considered.

tion being insufficient. The yearly endemic prevalence of typhoid in certain cities (Hamburg, Munich, St. Petersburg, etc.) is explained by the condition of the water in the conduits; it has already been frequently shown that typhoid can be almost completely checked by improvement in that direction. Not infrequently the transmission of the typhoid bacilli from water occurs indirectly—as, for example, by means of ice, artificial mineral waters, washed vegetables and other food stuffs, and, above all, by watered milk, which is an especially good culture medium for typhoid bacilli.

Direct contagion of typhoid through the air does not exist. Although its importance is not to be overestimated, there is a certain amount of danger of transmission of the disease from a typhoid patient to his environment. Nurses, physicians, and, in hospitals, other patients in the same ward, not infrequently become infected by soiling of the hands with stools or urine, kitchen utensils, linens, etc. The more unfavorable the surroundings (overcrowded neighborhoods, filth) the more readily can such types of contact-infection occur. The isolation of the typhoid patient is therefore desirable under any circumstances. Physicians may also become infected by post-mortem examination of typhoid cases.

[It is not probable that sewer gas in itself is an exciting cause of typhoid fever. Especially in large cities typhoid dejections are constantly finding their way into the sewers, which afford all the conditions favorable to the further growth and development of the poison. If, then, the drainage of any house is defective, the seeds of the disease can readily gain access to the interior of the house and infect susceptible individuals.

One of the most instructive epidemics on record is that in Plymouth, Pa., a town of eight thousand inhabitants. In the spring of 1885 a disease, at first supposed to be of a strange character, broke out in the place, and, before it ceased, affected twelve hundred persons, causing one hundred and thirty deaths. It was soon found that the malady was typhoid fever, which arose from one case, briefly in this wise: In January, February, and March there was a case of typhoid in a house on a hill sloping toward a water supply of the town. The dejections were thrown out on the snow, under which the ground was deeply frozen. On March 25th a sudden and great thaw occurred, the water did not sink into the ground, but ran immediately into the natural surface channels, and on April 10th the epidemic began. There were reasons, which it is not necessary here to detail, why the above source of water supply was drawn upon to an unusual degree just at that time, but it has been shown that those who derived their water from other sources were spared by the disease. The original case came from Philadelphia.]

In almost all cases the intestine seems to be the actual point of entrance for the typhoid poison into the human system. This is shown by the fact that in all cases which come to autopsy in early stages of the disease, the typhoid bacilli are mainly confined to the lymphatic tissues of the intestine. The typhoid poison is swallowed, either directly with water or polluted food, or after being in some other way introduced into the mouth. [Raw oysters grown in impure waters may convey the infection.—V.] If not destroyed in the stomach, it passes on in a viable state into the alkaline contents of the intestine, and here finds the conditions essential to its further development. It penetrates at first into the lymphatic follicles and Peyer's patches, and

thence goes on into the mesenteric glands, the blood current, the spleen, and other organs.

As in the case of most other infectious diseases, the occurrence of infection in typhoid is dependent not only on outward conditions, but also on an individual predisposition, namely, a varying individual power of destroying the poison that has entered the organism before any harmful effects of the poison have resulted. Even in the worst typhoid centers, where the possibility of infection must be universal, many escape the disease.

Age has an indubitable influence upon the liability to the disease. Typhoid is especially a disease of youthful, vigorous individuals, of fifteen to thirty years. Above that age it is much less frequent, although cases do occur at sixty and even seventy years. Formerly it was often said that young children were never attacked; but this was because the disease was not recognized, for in reality it is only children under one year old who seem to be seldom infected. At a later age, cases are by no means rare.

Sex cannot be shown with certainty to have an especial predisposing influence upon the frequency of typhoid fever.

Mental excitement and gross errors in diet seem to predispose to the disease. On the other hand, a certain immunity has been alleged to be given by many circumstances, especially pregnancy, the puerperal state, and other diseases already existing (tuberculosis, heart disease). Most of these statements are shown, however, by more extended experience, to be very doubtful. It is certain, however, that the occurrence of typhoid fever gives very probable though not absolute immunity against any later new attack. This established fact of immunity, not only for typhoid fever but also for several other infectious diseases, has till recently been empirical. At the present time Buchner, Behring, Ehrlich, Pfeiffer, and numerous others have thrown considerable light on the problems of natural and artificial immunity, and on the presence of specific antibodies in blood serum and tissues (antitoxins, bacteriolysins, agglutinins, etc.).

Finally, it must be mentioned that the necessary conditions for an abundant development and conveyance of the typhoid germs are beyond doubt dependent on the season. According to statistics, most of the typhoid epidemics come in the months from August to November, while generally the number of cases greatly diminishes from December to spring.

General Course of the Disease.—Extended experience shows that, after infection with the typhoid poison has taken place, a certain time must elapse before the symptoms of the disease appear. The length of this time, the "stage of incubation," is, unlike that of many other infectious diseases, not perfectly definite. On the average, it lasts two to three weeks, sometimes less time, sometimes longer. During this period the patient either feels perfectly well, or has certain slight symptoms, to which he pays more or less attention, according to his individual susceptibility. These prodromata consist of languor, disinclination to exertion, anorexia, slight headache, pain in the limbs, etc. Often they last only a few days. Not infrequently the patients state afterwards that they had felt the disease coming on for weeks.

The transition of the prodromata into the regular disease takes place sometimes so gradually that it is utterly impossible to take any one day as the first of the illness, in order to reckon from it its duration. It is usually, however, the first symptoms of a high temperature, chilliness, feverishness,

and the accompanying increase in general discomfort, which allow one to fix, with at least some accuracy, the beginning of the disease. A decided initial rigor is exceptional. After the fever begins, most patients soon take to their beds, although it happens often enough that the sick feel either unable or unwilling to give up, and keep on at work for days!

There have been manifold attempts to divide the whole course of the disease into separate periods. The most natural division seems to be into the three stages of development, height or fastigium, and decline (*stadium incrementi, s. acmes, s. decrementi*). Usually, however, physicians reckon according to the week of the disease. The first week corresponds to the developmental stage, the second, and in all severer cases the third as well, to the fastigium, the fourth (in light cases the third) to the decline. The following brief survey of the course of the disease holds only for the fully developed cases. It is to be noted that in individual epidemics there may be peculiarities in connection with the severity of the course of the disease, the development of certain complications, relapses, etc.

In the first week, the initial period, the general symptoms augment rapidly. The patients become, in severe cases, very languid and feeble, have generally an intense frontal headache, and complete anorexia, with great thirst. The fever, which is all the time gradually rising, is recognizable subjectively by alternating sensations of heat and cold, and objectively by the hot, dry skin, the parched lips, and the dry and coated tongue. The sleep is disturbed. For the most part there are no prominent thoracic symptoms, except that at times there is a sense of oppression in the chest, or some cough. The pulse is decidedly, but generally not greatly, accelerated, sometimes even now dicrotic. There is often a temporary epistaxis. The belly is not much swollen, as a rule, and but little if at all tender. However, the patients sometimes complain of abdominal pain as well as of backache. There is generally constipation. Usually the spleen, even at this time, exhibits a swelling that can be easily demonstrated.

Generally the fastigium has begun before the end of the first week. The severe general symptoms persist or even increase. The fever maintains constantly a considerable elevation. The patients become more stupid. Often delirium appears, especially at night. In the lungs there is developed a more or less intense and extensive bronchitis. The abdomen becomes more swollen. On the skin of the trunk appear, generally at the beginning of the second week, a number of small, pale-red spots, roseolæ. Instead of constipation, there is a moderate diarrhea. There are daily about two to four soft, thin, bright-yellow dejections.

The third week, during which in the severe cases the symptoms already mentioned persist, is the chief time of the numerous complications and of especial clinical events, about which we shall speak below at length. If the disease takes a favorable course, there comes at the end of the third week a decline of the fever; and then the general symptoms also improve, as a rule. The mind becomes clearer, the patient sleeps better, and gains some appetite. The pulmonary and digestive symptoms abate; convalescence gradually begins.

We will begin the presentation of the chief peculiarities by speaking of the course of the fever.

Course of the Fever.—Observation of the temperature in typhoid is so absolutely essential for the estimation of each individual case that no scientific physician ought to treat a case without regular measurement of the temperature. The measurements should be taken, if possible, in the rectum. Their frequency must, of course, be modified by circumstances, but it will probably be possible to have three or four measurements daily. At night, especially if the patients are asleep, it is generally not requisite to take the temperature. A general idea of the course of the fever can be gained only by representing the separate measurements graphically in a continuous "temperature curve."

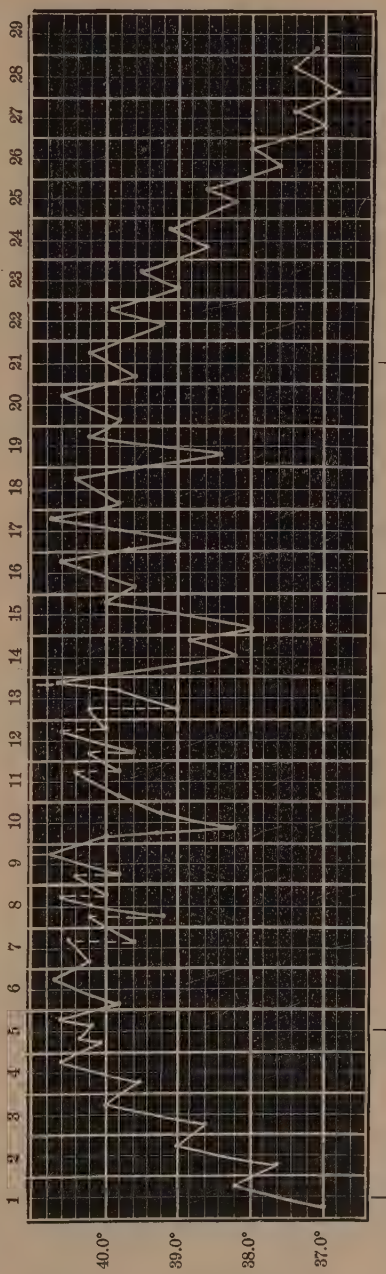

FIG. 3.—Temperatures in typhoid fever.—The dotted lines represent the remissions in temperature after cool baths, the crosses (+) the exhibition of antipyretics.

The typical curve of typhoid fever (see Fig. 3) falls naturally into three or four divisions. The first division is the initial period, or the pyrogenetic stage, and is seldom observed, since at this time the patients are generally not yet under the doctor's care. The initial period of the fever lasts, as a rule, some three or four days, seldom longer; and during this time the temperature rises, generally by gradual steps, so that the morning as well as the evening temperature is each day about 2° or 3° F. (1° to 1.5° C.) higher than on the day before. A sudden and considerable rise of temperature, such as occurs in many other diseases, is very rarely seen in the beginning of typhoid fever.

The second division of the curve represents the so-called fastigium, and corresponds to the height of the disease. During this time the fever presents, in most of the severer cases, the general character of "*febris continua*"—i. e., the spontaneous remissions of the fever seldom exceed 2° F. (1° C.). Almost always the lower temperatures come in the morning hours, and the

higher in the evening. In cases of average severity the morning remissions touch 102° to 103° F. (39° to 39.5° C.), and the evening exacerbations 104° to 105° F. (40° to 40.5° C.). Temperatures which reach or exceed 106° F. (41° C.) are seen only in very severe cases. Considerable morning remissions are always a favorable symptom, while morning temperatures of 104° F. (40° C.) or higher generally show the case to be severe. The duration of the fastigium varies with the severity and obstinacy of the case. It may last only a few days or one and a half to two weeks; in violent cases still longer.

In many cases of slight or average severity the period of decline follows directly on the fastigium; but in severe cases there frequently intervenes another stage, which Wunderlich has graphically named the "ambiguous" period. The temperature curve becomes irregular and more variable. The morning remissions may be great, even reaching the normal, while the evening temperatures are often still very high. This stage has accordingly been termed the "period of the steep curves." It may be said that in general the longer a case of typhoid lasts the more irregular will be the course of the fever.

The last stage—i. e., in cases of slight or average severity the third stage, and in severe cases commonly the fourth—is the period of defervescence or recovery. The peculiarity of this period in typhoid fever is that the fall of the fever is never by crisis, but always gradually, by lysis. Commonly the temperature descends by degrees, so that on each new day the morning remissions as well as the evening exacerbations are 1° to 2° F. (0.5° to 1° C.) lower. The zigzag form of curve, in which there are, of course, very frequently slight irregularities, must be taken as the rule. The duration of the defervescence generally exceeds that of the initial period. It lasts five to eight days, often longer. It is not very seldom that in defervescence the morning remissions become from the first very marked, even reaching the normal temperature, while the evening exacerbations become daily less and less, until they, too, are not above the normal. A third form of decline is much less frequent, in which the morning remissions become every day greater, while the evening temperature persists for some days at about the same height. Several times we have seen the fever take on a tertian type during recovery.

To this outline must be added a number of observations of practical importance.

The initial period does not exhibit especial variations from the course we have stated. Its entire duration is bounded by certain relatively narrow limits.

The fastigium presents, as already mentioned, the greatest varieties in its duration. In light cases it is wholly wanting, so that these consist only of a period of gradually rising fever, and of a gradual defervescence almost immediately consecutive to the rise. The entire duration of such light cases is only one and a half to two weeks. In other and tolerably frequent cases, which are often tedious, but still for the most part are light, the fever is not continuous, but remittent. The difference between the morning and evening temperatures amounts to 3° or 4° F. (1.5° to 2° C.), but the absolute height of the temperature is often not very considerable, so that the temperature curve at first leads to error and excites suspicion, for example, of tuberculosis. We have seen in Leipsic a number of cases in which the fever was perfectly intermittent during almost the entire illness, and for two to three weeks after-

noon elevations reaching 104° F. (40° C.) or more daily succeeded normal morning temperatures. These cases had the general course of light attacks.

Various influences, not to speak of therapeutic interference, may produce a considerable temporary remission of temperature in the course of the fastigium. Such a remission sometimes occurs spontaneously from the seventh to the tenth day of the disease. If a marked intestinal hemorrhage happens (*vide infra*), the temperature generally falls several degrees centigrade, and the less frequent instances of severe epistaxis have the same effect. If, in female patients, abortion or premature delivery occurs, we often observe a similar considerable fall of temperature, even without severe attendant hemorrhage. Perforation of the intestine often causes the temperature to fall rapidly. At times the occurrence of mental disturbances effects a moderate though noticeable lowering of temperature. Those great and sudden depressions of temperature remain to be mentioned which are accompanied by a very small but exceedingly rapid pulse and general prostration. Every such collapse, if severe, is a most dangerous event, and demands prompt and energetic medical treatment (*vide infra*).

The development of local complications, such as pneumonia, inflammation of the parotid gland or of the middle ear, etc., is generally accompanied by a considerable rise of temperature. The fever in such cases often becomes more irregular. This indicates the great practical value of thermometry. Almost every new rise of temperature or any considerable change in the ordinary course of the temperature has its special cause, and is, therefore, a warning to the attending physician to be vigilant. The cause of the change in the temperature is often not apparent until two or three days later.

The period of defervescence departs most frequently from its typical behavior by being lengthened out into a "stage of retardation." The morning temperature is then generally normal, while in the evening slight or moderate elevations continue. The reason for this long continuance of the fever may frequently be found in some not yet completely healed local complication, but often no such lesion can be demonstrated. Then we are commonly inclined to surmise sluggish intestinal ulcers which will not heal, or trouble in the mesenteric glands, etc. This slow fever may continue for weeks. It is prone to follow severe cases, but lighter attacks, especially in elderly or feeble patients, may also take on this sluggish character at a relatively early period. To these last-mentioned cases must finally be added a few others in which, during the whole course of typhoid fever, no febrile temperature at all, or only a very slight rise, can be detected.

Entrance into complete convalescence is shown with far greater certainty by the absence of elevations of temperature than by any other single symptom. There sometimes come, however, temporary elevations of temperature during convalescence, following some error in diet, long-continued constipation, or mental excitement. In other cases the new fever depends on some local sequelæ, e. g., a boil or a glandular abscess. Often, however, the most accurate investigation fails to demonstrate a cause. Especially in the beginning of convalescence there sometimes comes a high fever, or even a rigor, which may recur several times, but which soon gives place to a normal temperature. Generally no definite cause for these brief but decided elevations of temperature can be pointed out. Perhaps we might consider the possi-

bility of some affection of the mesenteric lymph-glands. These sudden and great elevations have seldom any grave significance.

This new fever which we have just described is best termed febrile recrudescence, or febrile sequela, in contrast with the proper typhoid relapse. That is, after typhoid fever has ended, the whole process may be repeated; and this occurrence is called a relapse. Particulars as to the behavior of the fever in such cases will be considered below, in connection with all the other peculiarities of typhoid relapses.

Phenomena and Complications Relating to the Separate Organs.[1]—Before we undertake a detailed discussion of the individual symptoms of typhoid fever, we must first make a brief general statement which is of very great significance for the correct understanding of almost every infectious disease. We include among the direct typhoidal symptoms all those morbid phenomena which are produced immediately by the typhoid bacillus itself or by its toxic chemical action; but, on the other hand, every patient sick with typhoid fever is exposed to many secondary infections (so-called mixed infections) from the intestinal ulcers, from the mouth, in the lungs, etc., whose invasion is rendered possible, or at least rendered easier, by the preëxisting typhoid infection. All the morbid symptoms arising in this way, which unite with the pure typhoidal symptoms in making up the whole picture of the disease, must, strictly speaking, be termed complications of typhoid. In the individual case it is often hard to decide whether a particular symptom is of a typhoidal nature or a complication. We must, however, hold fast to the essential difference between these two kinds of morbid phenomena, if we would obtain a deep insight into the nature and origin of the whole course of the disease.

1. Digestive Organs, Mouth, Ears, Parotid.—We think it best to begin our consideration of the more special symptoms with the phenomena referable to the intestinal canal, for the reason that the anatomical changes in the intestine are pathognomonic. Indeed, these alterations may sometimes become of surpassing import in a clinical point of view, although in the majority of cases the intestinal symptoms are clinically not nearly so prominent as the general symptoms that result from the infection of the system as a whole.

The characteristic typhoid lesion of the intestine consists of an affection of Peyer's patches, most marked in the lower part of the ileum. In the first week the patches swell gradually (stage of medullary infiltration). The rest of the mucous membrane exhibits at the same time more or less marked symptoms of simple catarrhal inflammation. In the second week, necrotic crusts form on the surface of the patches, which are cast off in the third week, leaving behind the typhoid ulcers. Toward the end of the third week the ulcers clean up, and then in the fourth week, if the case takes a favorable course, the ulcers heal. Smooth scars are formed, often diffusely pigmented. Experience shows that these scarcely ever lead to stricture of the intestine. The same process also goes on in a greater or less number of the solitary follicles as well as in the Peyer's patches themselves. We may add

[1] To avoid repetition, we have in what follows united a description of the anatomical changes with the presentation of the clinical symptoms.

that probably in lighter cases of typhoid (*vide infra*) there is often no actual ulceration. The swelling of the lymphatic tissue subsides in this case before sloughing occurs. We have already mentioned the occurrence of typhoid bacilli in Peyer's patches and the intestinal follicles.

The number and size of the ulcers formed have no direct relation whatever to the severity of the case. Although very extensive lesions in the intestine are often found in cases that end fatally, yet, on the other hand, we observe fatal cases in which only a few ulcers are found in the intestine. In cases with extensive intestinal lesions we often see follicular ulcers in the colon as well as in the small intestine (colo-typhoid).

The clinical symptoms referable to the intestinal canal are, as we have said, prominent only in exceptional cases. In the beginning of typhoid fever there is usually constipation. This may last throughout the illness, so that the patients have but one dejection in every two or three days, or often none at all unless an enema be given. As a rule, a moderate diarrhea begins during the second week. There are two to four stools, or sometimes more, each day. These usually have a characteristic bright-yellow color. On standing, they divide into an upper, cloudy, and quite liquid layer, and a lower layer composed of yellow, crumbly masses. They have generally an alkaline reaction, and upon microscopic examination they are found to contain, besides remnants of the ingesta and granular detritus, a few epithelial cells, round cells, many crystals of triple phosphate, and numberless bacteria. Pfeiffer and other investigators have been able frequently, although not invariably, to demonstrate the true typhoid bacilli in the dejecta by means of special methods of cultivation.

Severe diarrhea (ten or more stools daily) is relatively infrequent. In some bad cases we have seen the stools take on a dysenteric character. The autopsy showed in these cases unusually severe lesions of the colon and a diphtheritic inflammation of its mucous membrane. They were probably secondary complications.

Gaseous distention affecting the intestine, and especially the colon, is very frequent; a slight but evident gaseous distention, with a fluctuating, "air-cushion" feeling, is a very valuable symptom in the diagnosis of typhoid fever, but, as a rule, the meteorism is moderate. Indeed, severe cases of typhoid are observed in which the abdomen always remains concave. Marked tympanites is always an unpleasant complication. We saw one case, which ended fatally, with very great tympanites, in which the lesions were almost exclusively in the colon, and it was the enormous distention of its entire length which had so swollen the abdomen. The gurgling noise that can often be produced by pressure in the ileocæcal region, even at the beginning of the disease, depends on the localized distention of the bowel in that region. It is to a certain degree characteristic, although, of course, of no great diagnostic importance. Abdominal pain is often entirely absent. Some patients, however, complain of abdominal pain during almost the entire illness. On pressure, the belly is generally somewhat sensitive, but the tenderness is seldom extreme. It is more apt to be marked when there is constipation. Often such tenderness is due to a participation of the peritoneum in the disease, even when there is no perforation (*vide infra*).

There still remain two symptoms of the greatest practical importance,

both of which have a direct connection with the intestinal lesions: they are intestinal hemorrhage and perforation.

Intestinal hemorrhages in the course of typhoid are almost always due to the erosion of the walls of blood vessels in connection with the formation and throwing off of the crusts of the ulcers. The hemorrhages occur, therefore, most frequently toward the end of the second and during the third week. The blood pours out into the intestine, and is passed with the stools. Its amount may be small, or it may reach to one or two pints, or even more. Its color is generally rather dark. The later discharges are generally tarry. Liebermeister states that he has observed intestinal hemorrhages in 7.3 per cent of typhoid patients, and Griesinger in 5.3 per cent. We have ourselves found, in earlier statistics from the medical wards in Leipsic, 45 intestinal hemorrhages in 472 cases, i. e., in 9.5 per cent. In individual epidemics the frequency varies greatly. It rose in one year, for example, to eighteen per cent.

Intestinal hemorrhage is always a grave symptom. Even slight hemorrhages deserve consideration, for they may be the precursors of severer ones. Yet intestinal hemorrhage, even if profuse, is not necessarily fatal. Of the above forty-five cases of typhoid with hemorrhage, twenty-six ended in complete recovery. In eight cases, death occurred as the immediate result of the loss of blood. Eleven ended fatally after a time.

After every considerable intestinal hemorrhage, the symptoms of general anæmia, often even of collapse, appear. The fall of the bodily temperature has been already mentioned. The hemorrhage has sometimes a favorable influence on severe cerebral symptoms, for consciousness succeeds to the previous stupor or delirium. Often the hemorrhage is directly followed by recovery from the disease.

Much more ominous than the intestinal hemorrhage is the occurrence of perforation, as a result of the breaking through of a typhoid ulcer into the abdominal cavity, because, almost without exception, this is followed by a purulent or even ichorous peritonitis. Peritonitis is never caused by the typhoid bacillus itself, but by pyogenic organisms (cocci, perhaps bacterium coli?) which enter the abdominal cavity with the contents of the intestine. The amount of fluid peritonitic exudation is, as a rule, not very great. The serous membrane is often found covered merely with a fibrino-purulent or purulent-hemorrhagic deposit. The occurrence of perforation is sometimes marked by a violent pain suddenly felt by the patient; but it may also, in severe cases associated with profound prostration, take place insidiously. The abdomen is generally (not always) greatly distended and very tender on pressure, so that, even in stupor, patients groan while being examined. If gas has entered through the opening into the peritoneal cavity, we often observe absence of the ordinary dullness over the liver; but this symptom is to be employed cautiously as a factor in diagnosis, for absence of hepatic dullness may also result from distended intestines lying in front of the liver. When perforation has occurred, the patient soon looks collapsed, with cheeks fallen in and sharp, cool nose. Frequent eructations and vomiting often follow. The pulse becomes small and very frequent. The temperature generally falls as the peritonitis begins, and later it usually undergoes great variations.

Perforation of the intestine occurs most frequently in the third or fourth week of the disease, and much oftener in men than in women. In sluggish

cases, however, we cannot be without apprehensions of it till a late period. The perforation generally takes place in a coil of the lower part of the small intestine, in the right side of the pelvis, seldom in the vermiform appendix or in the colon. With few exceptions, death comes quickly, after a few days at latest. Only when the perforation is small and the intestines have become agglutinated at the onset is the course of the peritonitis slower, so that the symptoms are less violent and cause death in a week or ten days. Out of fifty-six fatal typhoid cases in the Leipsic medical clinic we lost five, or nine per cent, from peritonitis following perforation. Here and there a case of recovery has been reported, probably resulting from a limitation of the peritonitis through speedy adhesion of the intestines. Regarding the possibility of operative cure, *vide infra.*

It should be mentioned here that sometimes in typhoid fever a local or general peritonitis may occur through direct extension of the process to the serous membrane without actual perforation. We have seen in one case, as the result of the peritonitic bands and false membranes, complete occlusion of the intestine (ileus), and death.

Swelling of the mesenteric lymph-glands (less often of the retro-peritoneal glands as well) is found in typhoid almost as constantly as the anatomical changes in the intestine. Sometimes they break down, i. e., suppurate. In cases that have passed through the disease we often find considerable deposits of lime in the glands. These changes have a certain clinical importance; for, as already mentioned, we may often venture to refer a more or less tedious recurrent febrile state which has no other demonstrable cause to this lesion of the mesenteric glands. In some rare cases a general peritonitis has been observed as a result of the bursting of a suppurating gland.

The swelling of the spleen (acute splenic tumor) is, in typhoid fever as well as in many other acute infectious diseases, one of the most constant symptoms. The enlargement of the spleen can often be demonstrated as early as the end of the first week, and is, therefore, of considerable diagnostic importance; but percussion of the spleen is sometimes decidedly difficult and deceptive in this disease because of the existence of tympanites. The surest demonstration of splenic enlargement is, therefore, always by means of palpation, which, after a little practice, gives a positive result in the majority of cases. Absence of splenic tumor in typhoid is most frequently observed in elderly patients. The spleen may diminish considerably in size after severe intestinal hemorrhage. Pain in the splenic region, resulting from tearing of the distended capsule, is comparatively rare. The splenic infarctions which sometimes occur may, in exceptional cases, prove the starting point of a peritonitis.

Hepatic symptoms are seldom seen in typhoid fever, except that there may be a moderate swelling of the liver. The anatomical changes of "parenchymatous degeneration," and the frequent formation in the liver of the small lymphomata which Wagner discovered, have no clinical significance. The bile secreted is generally pale and scanty. This is a partial explanation of the light color of the stools. The frequent presence of typhoid bacilli in the bile has been noted above. A very rare complication, which we ourselves observed in one case, is acute yellow atrophy of the liver.

The stomach presents no especial anatomical changes in typhoid. Ano-

rexia is an almost invariable symptom in the beginning and during the course of all severer cases. There is seldom any desire for food till recovery begins; but then, if convalescence is undisturbed, the appetite soon attains an enviable keenness. Vomiting in the beginning or course of the disease is an exception, unless after some error in diet. We have already mentioned it as a symptom of peritonitis. Persistent singultus is a truly tormenting symptom that has been repeatedly observed by us.

The changes in the mouth and throat of typhoid patients deserve the careful attention of the physician. The lips and tongue are in severe cases dry and fissured. The lips are often covered with dry, black crusts, sometimes described as a "fuliginous coating." The tongue is apt to be thickly coated at first, but later cleans off from the edges and tip. In severe cases, especially if the mouth is not properly cleansed, a rather severe stomatitis may occur and produce superficial ulceration of the buccal mucous membrane and of the edges of the tongue. The gums sometimes become spongy, and are apt to bleed, as if scorbutic.

Actual sore throat is in general rare. The difficulty in swallowing, often complained of by patients, is generally due to dryness of the pharynx. In certain epidemics, however, the occurrence of sore throat at the beginning of the illness has been frequently observed. It may even happen that this early sore throat is accompanied by an erythema diffused over the body, so that at first suspicions of scarlet fever arise. The cases are very interesting and quite rare (although we have repeatedly seen them) in which disturbances of swallowing exist with the general typhoid symptoms from the beginning. On examining the fauces we see on the tonsils peculiar white, slightly elevated spots, which later form superficial ulcers. After a time these places heal, and in other respects the disease pursues a normal course. The suspicion is justified that in these cases there is a specific typhoidal disease of the tonsils (due to the typhoid bacillus itself), and such cases are called tonsillar or pharyngeal typhoid (analogous to the laryngeal typhoid, pneumo-typhoid, and nephro-typhoid, to be mentioned later). In such cases the typhoid bacilli have probably attacked the tonsils at their first invasion. It should also be mentioned that in severe cases there is often an extensive growth of thrush in the mouth and throat, and this may spread quite a distance down the œsophagus.

The changes in the mouth and throat are of especial interest, for the reason that they may be directly propagated to important neighboring organs. Starting from the pharyngeal cavity, the pathogenic agent, probably in most cases the staphylococcus, may penetrate through the Eustachian tube into the middle ear. Thus arise those inflammations of the middle ear which are not very rare in severe cases of typhoid, and which lead to perforation of the membrana tympani and to purulent discharges from the ear. The not infrequent inflammation of the parotid gland is also, as we believe, occasioned in a similar way, the inflammatory agent (sometimes the typhoid bacilli themselves) reaching the parotid gland from the mouth by way of Steno's duct. We do not regard the otitis and parotitis as especial localizations of the typhoid poison, but as secondary diseases for the occurrence of which typhoid fever merely furnishes the occasion, as when the mouth is imperfectly cleansed.

Purulent otitis in typhoid fever can easily be overlooked at first, since stuporous patients only rarely complain of pain in the ear or of deafness.

In addition, it is to be here observed that the difficulty in hearing does not always depend on middle-ear disease. We have seen several cases of almost complete (transient) deafness, combined with marked tinnitus, for which absolutely no cause could be found on examination with an ear speculum; in all probability there was some disease of the inner ear or of the auditory nerve. The parotitis appears most frequently in the third week, and generally on one side, though sometimes on both. It almost always becomes purulent, and discharges either externally or into the external auditory meatus, unless there is a timely incision.

2. Organs of Respiration.—Affections of the lungs are among the most frequent and important complications of typhoid fever, but are for the most part not a direct result of the typhoid infection, but pure complications. The bronchitis very often found in severe cases, and especially in patients who do not come till late under proper care, certainly is dependent on the imperfect expectoration of the bronchial secretions and on the inhalation of inflammatory agents coming from the mouth and throat.

Numerous cases of typhoid of slight or average severity, under proper care, run their course without any considerable bronchitis. In many other cases, and even severe ones, the bronchitis remains within moderate bounds, especially if the patient is brought promptly under proper care and treatment; but in severe cases, when marked disturbances of the nervous system arise, and the patient in his stupor expectorates little, swallows things the wrong way, and lies all the time on his back, passive and collapsed, the occurrence of a severe, diffuse bronchitis, especially in the lower lobes of the lungs, can hardly be avoided. Nor in such cases is there generally a mere bronchitis, but a more or less extensive catarrhal, lobular pneumonia, to be classed therefore under the so-called inhalation pneumonias (*cf.* chapter on lobular pneumonia). What was formerly termed "hypostatic pneumonia" is also almost invariably to be put in this group.

From the way in which these pulmonary disorders arise, we can understand why the bronchitis sometimes takes on a putrid character, and why the lobular infiltrations are, in severe cases, transformed into genuine gangrene. If such spots touch the pleura, they occasion the development of a pleurisy which is almost always purulent. In rare cases, pneumothorax may arise as a sequel to the perforation of a gangrenous infiltration into the pleural cavity. Various circumstances promote the occurrence of pulmonary symptoms. Thus we find it especially easy for a severe bronchitis and its sequelæ to be developed, in the case of elderly persons, or the kyphoskoliotic, or the corpulent, or patients who have previously suffered from emphysema or cardiac disease.

The subjective thoracic symptoms, in typhoid patients who have pulmonary complications, are generally not very prominent. It is only occasionally that patients complain in the early stages of typhoid fever of pain, and of a sense of oppression in the chest, or of cough, or of a stitch in the side; and even when such symptoms exist, the physical examination may give comparatively insignificant results. The severer pulmonary complications are seen mainly in those whose intelligence is more or less blunted, and who, therefore, make little complaint, are not much disturbed by the dyspnœa, and cough and expectorate little. A careful physical examination alone can enlighten us as to their condition. On auscultation, sibilant rhonchi are the chief signs observed in the

milder cases. In the severer ones there are moist, fine, and coarse râles, especially numerous toward the base of the chest. If there are abundant moist râles, we may infer that there is a lobular pneumonia, although this cannot be demonstrated with certainty till the separate islets of infiltration unite into a more extensive solidification, so as to afford dullness on percussion.

Röntgen-ray examination affords valuable information as to the condition of the lungs; for evident reasons, however, such an examination can only rarely and with difficulty be made.

In addition to the pulmonary lesions already mentioned, genuine croupous or lobular pneumonia does occur in typhoid fever. This is often an actual complication, dependent upon a secondary infection with the genuine pneumonia diplococcus. Such a pneumonia may come on early or sometimes during convalescence, and affect both the upper and lower lobes. There is probably also a true typhoid pneumonia caused by the entrance of typhoid bacilli into the lungs. Such a pneumonia cannot at present be recognized anatomically, but only by bacteriological investigation. Especial interest attaches to those cases of typhoid fever which begin with a lobar pneumonia. Often there is at first not the slightest suspicion of a typhoid fever, for the disease is regarded as an ordinary croupous pneumonia; but it is usually to be noticed that the illness does not begin suddenly with a rigor, but more gradually, and that from its incipiency the constitutional symptoms, the headache and splenic tumor, are more prominent than is usually the case in pneumonia. At the end of the first week's illness there is no crisis, but persistent fever. Now the pulmonary symptoms often retreat more and more to the background, while, on the contrary, diarrhea and rose-spots appear. The spleen is enlarged. In short, the clinical picture of typhoid is developed. It is not unnatural to suppose, although there is yet no absolute proof of the fact, that in these cases, which are fittingly termed "pneumo-typhoid," the infection with the typhoid bacilli has taken place exceptionally in the pulmonary tissue, and that, therefore, the first pathological changes are developed in the lungs.

Laryngeal Lesions.—The same causes which produce the bronchitis result also in a simple catarrhal laryngitis, with hoarseness. This is in severe cases accompanied by superficial ulcers on the vocal cords or the posterior wall of the larynx. Sometimes, again, the lesion is due to mechanical causes, constituting the so-called "*decubitus laryngis.*" The disorders which attack the less superficial structures of the larynx are fortunately rare. Chief among them is a laryngeal perichondritis affecting the arytenoid cartilages. This complication is justly regarded as of bad omen, and may lead to the rapid development of œdema of the glottis, with great laryngeal obstruction and threatening suffocation. These severe laryngeal affections in typhoid are regarded by some authorities, especially by Klebs, as always the direct effect of the infecting poison; but in most cases they are probably due to an invasion of staphylococci or some similar microbes. We have several times seen croup in typhoid fever, and it is a very dangerous symptom. In regard to their origin most of these mild and severe laryngeal affections in the course of typhoid fever are to be regarded as secondary inflammations; but specific typhoid disease of the larynx also seems to exist. The cases are of interest where the whole morbid process begins with a severe laryngitis ("laryngeal

typhoid") and is followed later by the ordinary symptoms of typhoid (*vide supra,* pharyngeal typhoid).

Among symptoms referable to the mucous membrane of the nose, epistaxis is important. It occurs in the beginning of typhoid with tolerable frequency, and is in one way not unfavorable, for it often mitigates the patient's headache. At a later period nosebleed may become a most unpleasant complication, as it is sometimes very difficult to check. We have even seen one fatal case due to persistent nosebleed. Other nasal symptoms are exceptional. There is an old saying that typhoid never begins with a coryza.

3. Nervous System.—The old term "nervous fever," which is still used by the laity, shows how frequent and severe are the nervous derangements which occur in typhoid. In cases of any severity there is almost always a certain dullness of intellect, often amounting to apathy and somnolence. The patients give monosyllabic and incomplete answers to all questions, and their statements about their previous history are often disordered and contradictory. There may even be sopor or a deep coma in the worst cases. All cases of this sort in which there was a condition of intellectual enfeeblement were termed by the old physicians "*febris nervosa stupida,*" in contrast to the "*febris nervosa versatilis,*" that form in which abnormal mental activity or delirium predominates. In severe cases delirium is very frequent. It is generally worse at night, and at times when the patient happens to be left alone. Very often he tries to leave his bed, because of his delusions, and talks of persons and things with which he was formerly familiar; or he is very noisy and restless, sometimes shrieking from groundless fears. We may add that these diverse nervous symptoms frequently succeed one another, or appear in combination. Sometimes a soporose patient may be heard softly whispering to himself in "muttering delirium."

Certain motor disturbances are often combined with considerable impairment of consciousness. There is a slight twitching of the muscles of the face and extremities. The old authorities gave the name *subsultus tendinum* to the sudden leaping into prominence of the sinews thus caused. It is best seen on the back of the hands. In severe cases the patient is sometimes observed to grind the teeth together; this is due to a cramplike condition of the muscles of mastication, and is justly regarded as ominous. We often see persistent tremor of the extremities and lower jaw; and it is especially in these cases, as we have demonstrated upon numerous patients, that the tendon reflexes and the mechanical excitability of the muscles are much increased. If deep coma comes on, the muscles become lax, the motions of the eye are not coordinated, and reflex excitability diminishes, or is wholly extinguished.

Headache is one of the most constant symptoms in the beginning of the disease. It is usually referred to the forehead or temples. The pain may be very violent, and sometimes takes on almost a neuralgic character. It almost always subsides in the second week.

If we seek the cause of these nervous symptoms, which are often so severe, we find that the anatomical changes in the nervous system, including the brain, bear no relation whatever to the severity of the symptoms observed during life. We sometimes meet with minute hemorrhages in the cerebral meninges, or meningeal opacity or edema, or a moist condition of the cerebral parenchyma; but the connection of these and similar changes with the symptoms

of the disease is often more than doubtful. Nor can the microscopic alterations in the brain, which have been reported, be regarded as important and authoritative. It is only in very rare cases that large cerebral hemorrhages or purulent meningitis have been found. As to this last, we should always be very cautious in making a diagnosis, as symptoms which would seem to be most conclusively meningeal—such as stiffness of the neck, rigidity of the whole spinal column, and occipital headache—may appear in typhoid patients, and yet the autopsy may show no trace of meningitis.

For a long time it was thought that the nervous symptoms were mainly a result of the fever; i. e., the effect of the overheated blood on the nervous system (Liebermeister and others). But this belief is not supported by unbiased clinical observation.

Although it is undeniable that elevated temperature has a harmful influence on the nervous system, yet in numerous cases there is no relation between the height of the fever and the severity of the nervous derangements. There are cases in which the fever remains continuously high for days, while the patient feels perfectly comfortable and presents no symptoms of any important cerebral disturbance. The opposite class of cases is still more numerous, in which from the very start there is always a low temperature, and, notwithstanding, the most severe nervous symptoms arise. Fräntzel and others have published very striking cases of this sort. Hence we must seek for some other special cause of the nervous symptoms, and according to our present views this cause should be sought in the intoxication from the toxins resulting from the infection. That the appearance of the nervous symptoms is dependent, not only on the material causes, but also on the susceptibility of the individual, is shown by the fact that certain patients are especially prone to exhibit marked nervous phenomena; for example, hard drinkers, "nervous" individuals, those who have suffered violent emotional disturbances shortly before the onset of the disease, etc.

The true psychoses most often take the form of mental confusion. The patients lose their orientation of time and of place as well as of persons and of the nature of their environment. By careful examination, disturbances of memory in reference to recent events and illusions of memory can readily be demonstrated. Occasionally, after delusions of a terrifying nature, states of violent excitement may ensue. In other cases the mental state takes on more of a depressive character.

We have repeatedly seen patients in such a state that they would lie almost motionless in bed, with eyes open, and perhaps assert that they were dead! In other cases there is mental excitement, sometimes combined with hallucinations, or there is confusion of ideas. In one case, in a girl who was evidently predisposed to nervous disorders, we saw typical hysterical insanity break out during the fever. Sometimes the mental excitement at the beginning of a relapse terminates in actual insanity. All these typhoid psychoses have, in general, a favorable prognosis. They end in complete cure after a number of weeks or at most after several months.

We have still to mention a number of nervous diseases that develop in the course of typhoid or after its decline. Neuralgia is sometimes seen, as well at the beginning as at the end of the disease. It is most frequent in the regions supplied by the trigeminus and the occipital nerves. Great hyperæsthesia of

the skin and muscles is not rare during convalescence. It attacks the lower extremities by preference. Paralysis of single muscles (e. g., of the serratus magnus), or paralysis of a single extremity, has been repeatedly observed as a sequela. The paralysis is generally of the atrophic variety, and is probably, as a rule, due to neuritis. Ataxia and spastic paralysis of the lower extremities are rare sequelæ. They probably depend on secondary myelitic disease.

Finally, there are sometimes developed, either in the course or at the conclusion of typhoid fever, the symptoms of a localized cerebral disorder (e. g., hemiplegia, aphasia, etc.), the anatomical cause of which varies. There may be a hemorrhage or an embolism, and probably in still other cases a localized encephalitis. Other nervous diseases (multiple sclerosis, for example) rarely follow typhoid.

4. Circulatory System.—Disturbances of the heart such as to produce striking anatomical changes are rare. The pericardium macroscopically almost always appears normal; the slight mitral or aortic endocarditis sometimes found has no clinical significance. The disturbances in the cardiac muscle seem more important. It is often more flabby than normal. The cavities, especially on the right side, are often dilated. With the naked eye we frequently see in the muscle itself cloudiness or fatty changes. Microscopic lesions are usually present and are much more pronounced. They consist ordinarily of a granular ("albuminoid") or more rarely of a fatty or hyaline degeneration of the fibers, and of genuine inflammatory foci of interstitial myocarditis (Hayem, Romberg). No marked disease of the cardiac ganglia has as yet been found.

The clinical significance of these changes should not, we believe, be overestimated. In all probability they are often without serious consequences and disappear completely with recovery from the disease. Sudden death (heart-failure?) occurs in typhoid, but it is very rare (see the chapter on diphtheria). Permanent disturbances of the heart after typhoid are also rare. When they occur they are perhaps due to the passage from an acute myocarditis to a chronic interstitial degeneration.

The pulse in typhoid is almost always accelerated, but in general it is true that in this particular disease the frequency of the pulse is not increased in proportion to the height of the temperature. On the average, the heart beats 90 to 100 times a minute. This comparatively low pulse rate occurs so often that it has some diagnostic value. When it remains at 140 or higher, in adults, it is always an unfavorable symptom. The more accelerated pulse rates generally occur with the onset of some complication. Temporary accelerations are easily produced by mental excitement or bodily exertion, as by sitting up in bed. In convalescence the rate is not infrequently subnormal.

Slight irregularities of the pulse are not rare, either in the acme or the decline of typhoid. Marked irregularity is always a grave symptom, although in many cases it passes off and the patient recovers.

Dicrotism, due to loss of tension in the wall of the artery, is so common that it is still regarded by elderly physicians as characteristic of typhoid, although it often occurs in the same way in other acute diseases. In many severe cases the height and strength and fullness of the pulse may cause no alarm, but often the pulse is notably weak and small. This is due not only

to the influence of the disease, but also to the previous condition of the individual (*vide infra*).

The diminished cardiac activity may result in venous thrombosis, especially in the lower extremities and in a crural vein. This sometimes causes swelling of one of the lower extremities during the later stages or convalescence. The swollen member generally regains its normal size after some weeks. In other cases the thrombosis occurs earlier, and in patients who are still too vigorous to suffer from cardiac weakness, so that we are forced to the conclusion that there is some local specific cause, a local thrombo-phlebitis due either to the typhoid bacillus itself, or, more probably, to the invasion of the walls of the vein by some secondary infection. A possible, but fortunately infrequent, result of these thrombi in the lower limbs is pulmonary embolism and sudden death.

In severe cases, which end in death, cardiac thrombi are sometimes found, with emboli in the lungs, spleen, kidneys, or other organs.

Œdema of the ankles and legs is very often seen in convalescents, especially when they first get out of bed. It is due to the weakness of the heart and changes in the vesicular walls. Once we saw a general dropsy develop at the end of a severe attack in a girl of fourteen. The autopsy disclosed no other possible cause for it than the extreme atrophy and flabbiness of the heart.

5. The Blood.—As in most febrile diseases associated with great emaciation, so in typhoid in severe cases the number of red blood corpuscles (and correspondingly the amount of hemoglobin in the blood) is much diminished. We found, for example, 2,800,000 to 3,200,000 in a cubic millimeter. In milder cases the figures do not differ materially from normal. It is a fact of greater interest and also of distinct diagnostic importance that it has been found (first by Halla, later confirmed by all subsequent observations) that in typhoid, in contrast with numerous other acute febrile diseases—pneumonia, erysipelas, sepsis, etc.—there is no persistent leucocytosis. On the other hand, following the transient leucocytosis of the first week, there is a pronounced leucopenia—i. e., a diminution in the number of leucocytes in the blood. The white blood cells drop to 5,000 to 3,000 per cubic millimeter, and in severe cases even lower. The decrease chiefly affects the neutrophile cells; however, the eosinophiles also almost entirely disappear. At the onset of the disease there is a marked diminution in the number of lymphocytes; they are greatly increased in the later stages. After the fever disappears, the leucocytic count rises rapidly, and now there is not infrequently observed a distinct lymphocytosis and an eosinophilia. In the presence of inflammatory complications the blood picture often changes—great increase in the neutrophiles. Concerning the presence of typhoid bacilli in the blood, see below.

6. The Skin.—The eruption seen in typhoid fever is characteristic and extremely important in diagnosis. The roseolæ or rose spots (light red, slightly elevated spots) appear at the beginning of the second week, usually on the trunk, and chiefly on the abdomen and back. The number varies greatly. Rarely they are entirely absent, most often in elderly persons. Sometimes they are very abundant, and extend to the thighs, the arms, and even to the neck and face. Often they vanish after a few days, but they may persist much longer. In the latter case they may become to a very slight degree petechial,

so that they will not entirely disappear on pressure. They often occur in successive crops. We have even seen several cases where new rose spots kept coming for some days after the fever had disappeared.

As to other cutaneous eruptions, we may mention first of all that herpes labialis is so rare in typhoid that in cases of doubtful diagnosis it is a factor in excluding that disease. Miliaria, urticaria, and superficial pustules are sometimes observed. Occasionally little bluish spots appear, especially on the trunk. These used to be called "*taches bleuâtres*" (pelioma typhosum); but later observations show that they are not connected with typhoid fever. They are due to pediculi. We might use the term pelioma typhosum to designate the kind of vesicles which we have repeatedly seen on the abdomen in severe cases. They are about the size of peas, and have sero-hemorrhagic contents. Boils and superficial abscesses are frequent, especially as disagreeable sequelæ in convalescence from severe attacks. There are often abscesses of the sweat glands in the skin of the axilla during convalescence. All these and similar cases of suppuration in typhoid fever do not depend, as a rule, upon the original cause of the disease, but upon secondary pathogenic germs, especially the staphylococcus, for whose entrance the typhoid process has merely prepared the way. Extensive ecchymoses are very rare, and are symptomatic of a general hemorrhagic diathesis. Petechiæ are frequent during recovery. They are generally seen in the follicles of the skin below the knee. There have been a few cases of gangrene in the lower extremities, especially in the toes. We saw in one patient an extensive gangrene of the skin of the abdomen, probably due to the ice bag employed.

Finally, we must mention that bedsores are prone to develop in severe or neglected cases. The localities most often attacked are the nates, the furrow between them, and the heels. A bedsore may be so extensive, and accompanied by such undermining of the skin, as to be a dangerous or even fatal complication.

The epidermis often scales off to a considerable extent during convalescence after a severe attack of typhoid. Everybody knows how the hair falls out after the fever, but it is sure to grow again. The nails also are not infrequently affected, becoming rough and brittle, or even falling off.

7. Muscles, Bones, Joints.—Zenker has discovered a degeneration of the voluntary muscles which occurs in typhoid as well as in other severe diseases. It is called the "granular" or "waxy" degeneration. Whether it has clinical symptoms cannot be determined. Perhaps it may explain the great muscular hyperæsthesia which is often observed, and the muscular pains, which may be very trying. Severe cases sometimes have hemorrhages into the muscles, particularly the rectus abdominis.

Lesions of the bones and joints occur but seldom, although other observers and we ourselves have repeatedly seen periostitis and osteomyelitis in the ribs, tibia, etc., following typhoid.

Quincke first described a typhoid spondylitis with secondary symptoms due to pressure on the nerve roots. Its causation was demonstrated by E. Fränkel, who found numerous small foci of typhoid bacilli surrounded by localized areas of necrosis in bone marrow, especially in that of the vertebral column. Perhaps the frequent bone pains of typhoid patients may be due to such foci. The occurrence of leucopenia in typhoid (*vide supra*) may also be due to these

changes in the bone marrow. The typhoid bacilli are almost always to be found in the larger, purulent, periostitic, or osteitic areas.

8. Genito-urinary Apparatus.—Genuine, acute, hemorrhagic nephritis is a very rare complication. It does occur, however, and has even given rise to the establishment of a special "renal form of typhoid fever" (nephro-typhoid). This name applies especially to those cases in which a severe acute nephritis is the predominant symptom at the start, while at a later period the course of the fever, the intestinal symptoms, the rose spots, etc., show the disease to be typhoid fever. Nephro-typhoid is analogous to pneumo-typhoid and tonsillo-typhoid. A simple so-called febrile albuminuria occurs very frequently at the acme of typhoid, and is not to be interpreted unfavorably. It is probably the result of that slight parenchymatous degeneration of the kidneys which occurs in typhoid with the same frequency as in most of the other severe infectious diseases. In the albuminous urine of typhoid patients the bacilli are almost invariably present. In other respects the urine in typhoid presents the same peculiarities as in most other severe febrile diseases: its amount is diminished; its color dark; its specific gravity increased. The rare presence of urates in the sediment is worthy of note; this may bear some relation to the leucopenia of typhoid (*vide supra*). After cessation of the fever there is often a marked polyuria (3 to 5 liters daily of a urine of a low specific gravity) that may last for weeks. It should be added that the urine often contains indican and at the height of the disease almost always gives Ehrlich's "diazo-reaction." The disappearance of the latter reaction indicates a favorable prognosis. If the reaction persists after the fever has disappeared, one may be prepared for a relapse. Cystitis occurs not infrequently toward the end of the illness; it is often a secondary complication (a catheter infection, for example), but may be a specific typhoid infection.

In men, orchitis is sometimes observed. Women often have their catamenia at the beginning of typhoid. Later in the course of the disease, and in convalescence from severe attacks, the menses are often absent for several periods. In pregnant women there is considerable danger of abortion or miscarriage.

Peculiarities in the Course of the Disease.—The above statements show an almost inexhaustible variety in the possible symptoms and complications of typhoid. The course of the disease as a whole may likewise present many diverse forms and peculiarities. We shall attempt merely to cite the most essential.

The numerous light and rudimentary attacks (*typhus levissimus*) are first to be mentioned. It was not recognized till lately that they belonged to typhoid fever at all (Griesinger). They used to have all sorts of names applied to them, the favorite term being "gastric fever." This light form lasts from eight to fourteen days. The fever is moderate and often decidedly remittent. There is almost no proper fastigium. The typhoid symptoms are but slightly developed. There is no severe pulmonary or cerebral disturbance. There is generally a moderate diarrhea, the spleen is plainly enlarged, and often rose spots can be found. The diagnosis of these cases is, of course, difficult in proportion to the scanty development of typhoid symptoms. It is best established by demonstrating an ætiological relation between these cases and others which are plainly typhoid fever. A positive diagnosis can only be made by bacteriological methods (*vide infra*).

Abortive typhoid is justly distinguished by Liebermeister from *typhus levis*. The name belongs to cases which begin with severe symptoms and high fever, as if they were going to be grave, but in which these violent symptoms disappear after a few days and give place to a rapid convalescence.

On the other hand, there are cases which for a long time cause so little subjective discomfort that the patient does not even go to bed (walking typhoid). It is not till quite late that there occurs a sudden change for the worse, or some severe complication. Thus it has happened that persons who were apparently healthy have suddenly had all the symptoms of a severe peritonitis due to perforation and have died, the autopsy disclosing the lesions of the third week of typhoid fever; or a mild typhoid assumes the walking type and is not detected, but later a severe or dangerous relapse may occur.

The individual circumstances are very important in weighing each case, for they may modify the disease in many ways.

In children it is a remarkable fact that typhoid ulcers are much less frequent than in adults. This explains why intestinal hemorrhages and peritonitis are much rarer in children. Marked cerebral symptoms are, on the other hand, very frequent. In severe cases children sometimes exhibit the peculiar symptom of a continuous penetrating screaming. In other, mild, cases the children are soporose.

In the aged the diagnosis of typhoid is often very difficult, since the course of the disease is frequently irregular. Generally the fever is not very high, and it very seldom exhibits distinctly the type described above. The pulmonary or cerebral symptoms predominate as a rule.

In the corpulent, typhoid fever is often very severe, so that our prognosis must always be rather grave, especially if pulmonary symptoms arise.

Hard drinkers are also in especial peril in this as in all other acute diseases. Dangerous cardiac weakness is prone to appear. Severe cerebral symptoms are frequent. It is, however, surprising that true delirium tremens is relatively infrequent, although so common in pneumonia.

The influence of previous strong mental excitement and of certain already existing diseases (cardiac disease, emphysema, kyphoskoliosis, etc.) has been already mentioned. Finally, we repeat that often the different epidemics present certain peculiarities. For instance, in one the type of the disease will be severe, in another mild. In one epidemic relapses are comparatively frequent, in another exceptional. The same is true with regard to the frequency of the appearance of certain symptoms, such as intestinal hemorrhage, perforation, pneumonia, or nephritis. Indeed, it has even been observed that those cases which occur during a given epidemic in the same family or house or block sometimes present striking resemblances to one another ("group typhoid" of E. Wagner and others).

Relapses of Typhoid Fever.—Typhoid fever exhibits in many cases the peculiarity of repeating itself completely after having run its entire course and disappeared. This process is called a relapse. It is in all probability the result, not of a fresh infection of the system from without, but of a renewed development (a new generation) of the germs already present. One must think, however, of the possibility of a fresh autoinfection. Errors in diet, psychical disturbances, etc., sometimes appear to cause the relapse; usually, however, the exciting cause cannot be discovered.

A typical relapse is like a first attack in all clinical and anatomical particulars, with this difference, that everything is more condensed, and lasts a shorter time than in the first attack. The interval between the two, during which there is no fever, lasts seven to ten days. It may be longer, and is often shorter. Sometimes the relapse follows immediately upon recovery. Indeed, it may even happen that, before the patient has completely recovered, his temperature begins to rise again in the characteristic steplike way. To such cases as this last the term recrudescence is applied. Except in the time of its beginning, it may be just the same as a genuine relapse. In the interval between the two attacks many persons are perfectly comfortable, and appear to be fully convalescent. There is often, however, a slight evening rise of temperature. It is noticeable that the splenic tumor does not completely disappear after the first attack in many cases which are followed by a relapse. The persistence of the "diazo-reaction" has already been referred to.

The relapse is generally briefer, as we have said, than the first attack, seldom lasting more than fifteen to eighteen days. The temperature rises more rapidly, perhaps in two or three days. The fastigium is shorter, the decline more abrupt. The absolute height of the temperature may be very considerable, even exceeding that in the first attack. Rose spots appear as soon as the third or fourth day. The stools become liquid, the spleen enlarges again, and all sorts of complications may arise. The danger occasioned by a relapse may, however, be overestimated. On the whole, a relapse is not so very dangerous, and it is especially noticeable that the subjective symptoms, such as headache, are often slight. A severe relapse may, however, follow both mild and severe attacks. In other instances the relapse may prove merely rudimentary.

The frequency of relapses varies considerably in different epidemics. In Leipsic we had relapses in about nine per cent of all cases, but in separate years the percentage varied between four and sixteen. Out of about five hundred cases we have seen three in which there were two successive and typical relapses.

Diagnosis.—The diagnosis of typhoid fever may be perfectly easy, but, if the case be anomalous, or come under observation at a late period, it may be extremely obscure. It depends, above all, on the purely clinical manifestations of the disease. Important factors are the gradual onset, then the height and course of the fever, with no demonstrable localized disease to account for the fever, and the rose spots. Less characteristic, but still of value, are the stools, the tympanites, and the swelling of the spleen. In doubtful cases, the persistent slow pulse, the absence of leucocytosis, and the "diazo-reaction" may aid in the diagnosis. Ætiological factors, such as the occurrence of undoubted cases of typhoid in the neighborhood, are of great diagnostic value in obscure cases. Sometimes the diagnosis cannot be established till the appearance of certain symptoms, like intestinal hemorrhage, a characteristic mode of convalescence—viz., by lysis—or a relapse. It is an important rule not to make a diagnosis of typhoid after a single examination. It is generally necessary to observe the case accurately for several days before the diagnosis can be established. The differential diagnosis from other acute diseases, such as miliary tuberculosis, acute endocarditis, meningitis, etc., will be considered in discussing these diseases.

The diagnosis is usually most difficult in the cases with an imperfect history which are first seen in a severe "typhoidal" state, with high fever, disturbances of consciousness, etc. These are the cases in which we must also consider, beside typhoid fever, miliary tuberculosis, acute septic or pyæmic infection (including acute malignant endocarditis), meningitis, severe "typhoid pneumonia," etc. The clinical differential diagnosis between typhoid and these conditions will be spoken of later.

It is just in these doubtful cases that the newer methods of bacteriological diagnosis render incalculable aid.

The bacteriological diagnosis of typhoid fever is arrived at by two means —first, the examination of the blood for specific properties; secondly, the direct examination of the blood for typhoid bacilli. The first method (serum diagnosis, Gruber-Widal reaction) is based on the principle that the typhoid infection (like many other infections) stimulates the development of specific "antibodies" in the blood serum of the infected patient. Pfeiffer was the first to discover that typhoid bacilli, injected in lethal amount into a rabbit's abdomen, become disintegrated when a small quantity of serum from an immunized animal is simultaneously added ("bacteriolysis"). Gruber found that the blood serum from patients recently recovered from typhoid had a peculiar grouping, "agglutinating" effect on living typhoid bacilli, and Widal was the first to show that this "agglutination" could frequently be observed during the course of the disease.

When the necessary precautions are taken, the Gruber-Widal reaction is of great practical importance. Although not absolutely diagnostic, it is of great weight both when present and when absent. The macroscopic and the microscopic reactions are to be differentiated. The former is carried out by adding one or more drops of the blood serum of the patient to a uniformly cloudy suspension of living typhoid bacilli in 5 c.c. of bouillon. The test tube is allowed to stand for a few hours (best in a thermostat at 37° C.). If the reaction is positive, the bacilli are agglutinated in a concentration of 1-to-100, or at least of 1-to-50. They become clumped, sink to the bottom, the bouillon becomes perfectly clear. Even more characteristic is the *microscopical* serum diagnosis. To one drop of a fresh bouillon culture of typhoid bacilli the serum of the typhoid suspect is added in varying proportions. A drop of the mixture is examined under the microscope. If the reaction is present, the evenly distributed motile rods become clumped and immotile in a few minutes or, at most, in half an hour (see Fig. 4). The blood serum of typhoid patients shows this peculiarity to so great a degree that one drop is sufficient to give the reaction with the typhoid bacilli in suspension in twenty, thirty, and even up to one hundred drops of bouillon. On the other hand, the serum of healthy individuals, or of those suffering from other diseases than typhoid, does not possess this agglutinating power at all, or only to a very much

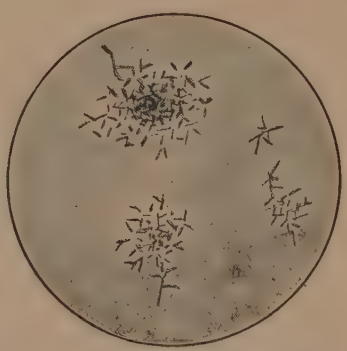

Fig. 4.—Widal reaction. Agglutination of the typhoid bacilli. (Compare with Fig. 2.)

slighter degree. Typhoid is thus almost invariably present if agglutination occurs with a 1-to-50 to a 1-to-100 dilution. The reaction is sometimes present on the third or fourth day of the disease, but, as a rule, not till the beginning of the second week. Very rarely, it takes place only at a later period. If there is no sign of agglutination in a case of fever of two weeks' duration, one may almost absolutely exclude typhoid fever. The reaction persists for a considerable time (months and even years) after the attack of the disease. The Gruber-Widal reaction can be obtained with the serum from vesicles or with serous inflammatory exudates of typhoid patients.

The significance of the Widal reaction is somewhat limited (R. Stern), because, on the one hand, it is not present in some cases of typhoid fever, and, on the other, the serum in other diseases (infections with colon bacilli, with B. proteus and icterus) may agglutinate the typhoid bacilli. Despite this, the Widal reaction, in combination with the clinical symptoms, remains of the utmost value in the diagnosis. Ficker's observation, that a suspension of killed and ground-up typhoid bacilli is agglutinated by typhoid serum, is of value in general practice. Ficker's "typhoid diagnosticator" (to be obtained from E. Merck, of Darmstadt) is based upon this possibility of a macroscopic examination without the use of any bacteriological apparatus.

The ultimate achievement of the bacteriological diagnosis of typhoid must, however, be the demonstration of the bacilli in the body of the patient. This can only be done, to be sure, in a bacteriological laboratory, and is therefore not available in general practice. But when it can possibly be performed, the direct proof of the presence of typhoid bacilli permits a certainty of diagnosis that is not possible by any other means. The search for the bacilli in the urine is, as yet, of the least diagnostic value. Of more importance is their demonstration in the feces. Since the introduction of the Drigalski-Conradi culture medium the differentiation between the typhoid and the colon bacilli has become a much simpler matter. The medium is an agar plate containing milk sugar, sodium carbonate, nutrose, and litmus. Most of the other varieties of bacteria are excluded by the addition of "crystal violet" to the medium, thus leaving behind the colon and typhoid colonies. The colon bacilli generate lactic acid, and therefore the colonies are red; the typhoid bacilli grow in blue colonies. The nature of the latter is established by further examination (agglutination, etc.). The bacteriological examination of the blood has, however, gained the greatest significance (Schottmüller). About 20 c.c. of blood are removed from one of the veins of the arm by means of a Luer syringe, and is at once mixed in several tubes with liquefied agar at a temperature of 45° C. (113° F.) and poured into Petri dishes, which are placed in the incubator at 37° C. (98.6° F.). In positive cases small gray colonies appear in the blood agar in twenty-four to forty-eight hours. The deeper colonies look black, owing to the formation of sulphid of iron. The deeper colonies are typhoidal and must be subjected to further microscopical examination. With careful technic the method gives excellent results. It is employed in all typhoid and typhoid-suspect cases in the Breslau clinic. The positive finding of typhoid bacilli in the blood occurs in at least ninety per cent of all cases of typhoid, and has made the positive diagnosis for us in many cases in which the diagnosis was otherwise uncertain or even impossible. Even in the first days of the fever the bacilli

can often be demonstrated in the blood. In fatal cases the bacilli in the blood increase greatly in number shortly before death. The attempts recently made to grow the bacilli from smaller quantities of blood (1-to-2 c.c.) are of great practical importance. For this purpose there must first be a preliminary "enrichment" of the bacilli. This is accomplished through the addition of sterile bile or pepton solution to the blood, and then placing the mixture for twenty-four hours in the incubator.

Prognosis.—A perfectly favorable prognosis should never be made. Cases which seem the mildest may become dangerous (from intestinal perforation, etc.). Yet, if there are good nursing and good treatment, typhoid fever is not a particularly dangerous disease, and we may hope for recovery even in very severe attacks. The danger lies, first, in the severity of the infection, as shown chiefly (though not wholly) by the height of the fever and the intensity of the general symptoms. A further danger is the appearance of the complications already enumerated and discussed. Thirdly, the constitution and condition of the individual are important. The circumstances coming under this head have likewise been repeatedly mentioned above. All these factors must be carefully estimated before we decide as to the danger in each case and make our prognosis.

The mortality in typhoid varies greatly in the separate epidemics. The severe cases are undoubtedly more frequent at some times than at others. This renders it difficult to give statistics which are universally applicable. We may in general reckon on an average mortality of about ten per cent, and measure the severity of separate epidemics by this standard. Numerous observers agree that the treatment now in vogue has decidedly diminished the mortality. It was formerly not rare for it to reach twenty or twenty-five per cent.

Treatment.—A specific cure for typhoid—i. e., some remedy to destroy the specific cause of the disease within the system, or to render it harmless—is as yet unknown. Bacteriologists are now attempting to discover a specific remedy, in the same way that they are attempting it in all of the other serious infectious diseases. An effort has been made to stimulate the formation of immune bodies by the injection of small quantities of killed typhoid bacilli into the typhoid patient. The injection of other kinds of bacteria (especially the Bacillus pyocyaneus) has also been tried. Recent investigations have dealt with a serum therapy, the immune sera being obtained from animals. All these studies are of great scientific interest, but their practical results are still very doubtful.

Under these circumstances the treatment of typhoid must still be chiefly dietetic and symptomatic, and in one sense prophylactic. We must fight the symptoms already present, and further seek, as far as possible, to defend the patient from the attack of certain dangerous secondary disorders. Starting out with this view, the proper treatment of typhoid fever is a task of the highest importance, and by no means a thankless one.

We will begin by considering the general treatment. The sick-room must not be too warm, and must be frequently and thoroughly aired. The sick-bed must be well cared for. If effort be made to prevent bedsores, we shall obviate one source of pain and danger (*vide supra*), and save ourselves and the nurse much trouble. Those who are very ill should therefore be laid on

an air cushion or, if possible, a water bed. The patient should be told not to lie always upon his back, but to change now and then upon his side. The back, the region of the sacrum, and the heels are to be often bathed with French brandy, etc., and anointed. The minutest bedsore is to be treated carefully. It should be cleansed twice a day and dressed with a mercurial ointment or one containing Peruvian balsam (1-to-30),[1] etc. If the bedsore be extensive, careful dusting with powdered dermatol, or a similarly acting dusting powder, is very efficient treatment. We should be particularly careful not to let the skin be undermined. If this has already occurred, we must be prompt in the use of the knife.

We cannot too strongly recommend that the mouth should be kept clean. In a light case the patient can see to this himself, but otherwise the mouth and tongue must be frequently cleansed with a linen cloth wet in cold water or a solution of borax (1-to-30). Perhaps we need hardly repeat the reason for this excessive cleanliness. It lies in the causative relation between stomatitis and inflammation of the parotid gland, and of the middle ear. If the tongue and lips be dry, they may be touched with glycerin.

The diet must be at once liquid and nourishing. Milk is excellent, and it should always be ordered, but it can, unfortunately, be taken by very few patients continuously. It is often better borne if coffee, tea, or a little brandy be added to it. Cocoa made with milk may also be given for a change. In severe cases we have frequently employed the various baby foods, such as hygiama, with benefit. Besides milk, the thick soups, gruels (oatmeal), soups containing sago, rice, farina, and the like, are to be ordered. Meat broths are also allowable, and may be made more nutritious by various additions (meat juice, sanatogen, nutrose, perhaps an egg). Sometimes calf's-foot jelly is of value. The demands that patients often make for more solid nourishment may be acceded to without risk by feeding them on rolls or zwieback that have been soaked. Cakes, sweet crackers, biscuits, and the like are also useful. When the strength of the patient is dangerously reduced, beef tea or expressed meat juice should be ordered. The newer artificially prepared meat juices and albuminous preparations may also be employed with benefit.

When the fever takes a sluggish course, we must often begin to give stronger nourishment before the fever has ended. The best drink is cold water, which must be offered to the patient even when he does not ask for it. Lemonade and similar preparations generally become distasteful in time. Drinks containing carbon dioxid are to be avoided, because they increase the meteorism. Cold tea with milk as well as with the fruit juices (especially the cranberry) is good. In severer cases we should give some good strong wine, such as port, Malaga, or Hungarian wine, but it is not necessary to force the patient to take wine if he does not care for it. If the patient desires beer, we need not hesitate to give it in moderate amount. During convalescence we should be very careful about diet, since errors often have disagreeable consequences. We must wait till there has been no fever at all for one to two weeks before we allow a solid, animal diet, and return by degrees to common sorts of food.

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[1] The unguentum balsami peruviani is made by mixing one part of balsam very thoroughly with thirty parts of the glycerite of starch (B. P.). It is not officinal in Germany.—Trans.

The general and dietetic treatment which we have thus far discussed is very important. Outside of this, it is our opinion that there is only one method of treatment to be chiefly considered—at least under the present limitations of our therapeutic ability. This method consists in the persistent use of cool baths, as first practiced by Brand in Stettin. Although the indications for this method of treatment are no longer exactly what its original promoter held them to be, and although, therefore, some of the minutiæ of the treatment must be changed, there is at present no other single method of treating typhoid fever which has so numerous and evident advantages for the patient when properly and moderately used. To carry it out in private practice may often be more difficult than in a well-appointed hospital. However, even in private houses it will generally be possible to manage it, and we regard it as the duty of every physician who undertakes to treat a severe case of typhoid to try his best to have the baths employed.

The great advantages of the treatment by baths are: 1. The baths diminish the fever, if their temperature be only sufficiently low, by direct absorption of heat. The baths thus obviate, as far as possible, all the bad effects which might result from a rise of temperature. 2. The direct influence of the baths upon the nervous system is still more important. The intellect becomes clearer, the apathy and dullness diminish. In fact, if baths be used, we do not see nearly so often as formerly the grave "typhoid condition." It is thus evident that bathing not only causes an improvement in the subjective sensations of the patient, but brings in its train many other beneficial effects. The patient takes his nourishment better, does not so often swallow the wrong way, coughs more effectively, is easier to move, and his body and his mouth can be better cleansed. 3. The influence of the baths upon the respiratory organs is of the greatest importance. We refer especially to the stimulation to deeper inspiration, and the promotion of expectoration. The best proof of the benefit of this influence is the circumstance that if patients are subjected to baths from the start, it is comparatively a rare thing for severe bronchitis, atelectasis, and catarrhal pneumonia to develop. 4. The good care of the skin, which the bathing makes possible, is not to be despised. Since this treatment has been introduced, bedsores are much rarer in typhoid than before. 5. Lastly, the baths are sometimes observed to have a diuretic effect.

What has been said shows that the height of the fever is by no means the sole indication for the employment of baths, at least in our opinion. The condition of the nervous system and of the respiratory organs is also to be considered. It is true that numerous mild cases run a favorable course without a single bath; but we should always remember that this treatment is not only directed against the symptoms already existing, but has also a prophylactic importance, since it tends to prevent any severe cerebral or pulmonary manifestations.

We will pass on to the special method of carrying out balneo-therapeutics in typhoid. Full baths are generally employed, immersing the patient to his neck. The tub must stand, if possible, by the bedside. In hospitals, where there are beds on rollers, it is a better way to wheel the patients into the bath-room. All who are severely ill should be lifted into the bath and there held and supported, to avoid any bodily fatigue. During the bath the skin should be gently rubbed. This averts unpleasant sensations of chilliness. The tem-

perature of the water should not be set too low, especially for the first baths. We begin at 85° to 90° F. (30° to 32° C.), or, if the individual be elderly or sensitive and timid, at even warmer temperatures. When the patient has become accustomed to the temperature of the water, we can cool off the bath still further. Baths below 73° F. (22° C.) have scarcely ever been used by us, and we believe that they are seldom needed. A very satisfactory average temperature is 80° to 85° F. (25° to 30° C.). A bath lasts, on the average, ten minutes. If the patient feels very cold or very uneasy in the bath, it must be cut short. After the bath the patient is at once lifted into bed, wrapped up in a sheet previously made ready, and wiped dry, with rather vigorous rubbing of the extremities and back. The moist sheet is then removed. The patient is covered up rather warmly, and is given some hot broth or a sip of good strong wine. The effect of the bath upon the temperature is measured about half an hour later by the rectum. If the temperature be 2° to 3° F. (1° to 2° C.) lower than before, the result is deemed satisfactory. Often the difference is greater, but in severe cases the fever may be so obstinate that the temperature remits only a small fraction of a degree. In such cases it is sometimes permissible to lower the temperature of the bath still more, or continue it a little longer. If cool baths are ill borne, protracted baths of lukewarm water are sometimes very efficient (Riess, and others).

In so far as the height of the fever furnishes an indication for baths, we may accept 103.6° F. (39.8° C.) in the rectum, as the temperature calling for a bath, but spontaneous variations of the temperature of the body are always to be regarded. In cases which show a low morning temperature without special interference, the height of the evening temperature is seldom an indication for a cold bath. We regard it as very important that the baths be not given too frequently, since their advantages may thus be overbalanced by their incontestable evil consequences. At present we rarely prescribe more than two to four baths a day. At night we have given baths only very seldom, when forced to by extremely high temperatures or other bad symptoms. It must be a mistake to wake a patient who is quietly sleeping, and put him into cold water, even if his temperature is above 104° F. (40° C.). On the other hand, even if the temperature be not excessive, or even if it be normal, there is, as we have said, no better remedy than the baths for severe pulmonary or cerebral symptoms. In such cases we often raise the temperature of the baths a little, and during them we have colder water poured upon the head and back. If we do this, the ears must be stopped with cotton wool, lest the cold water find its way into them.

Advantageous as the bath treatment of typhoid generally is, it must, however, like every other therapeutic method, be used with wise moderation and with constant regard to individual conditions. If the patients are very weak, show a strong aversion to the baths, or feel dull and exhausted instead of refreshed after them, it is well to consider whether we really do good by continuing their employment. In such cases we have often advised cold wet packs of the whole body in bed, instead of the baths, and we consider such a use of cold as very advisable in these cases. The antithermic action of the pack is, of course, much less than that of the bath, but it can easily be aided by the exhibition of antipyretics internally. The respiration and the nervous system, however, are almost always most favorably affected. Most patients readily consent

to the packs and lie quiet for an hour or two in the wet envelope. We would urgently recommend the more frequent use of packs, especially in private practice, where the bath treatment often meets with greater difficulties than in hospitals. We should here always begin with higher temperatures, and only gradually go on to cold packs.

There are a number of contraindications to the use of baths which cannot be disregarded. First, the occurrence of intestinal hemorrhage, however slight, and likewise the suspicion that peritonitis is developing, prohibit bathing. In these cases quiet is the very first requirement of the patient, and the baths must be at once discontinued. The onset of otitis, severe laryngeal affections, and acute nephritis are also complications in which we should properly omit the baths entirely or employ them very cautiously, giving them warmer and less frequently. Sometimes rheumatic pains in the limbs or an attack of boils render the continued use of baths difficult. In short, we see that nothing could be more foolish than to try to establish a general rule for the bath treatment of typhoid.

In many cases of typhoid fever any medication, in addition to dietary and hydrotherapeutic measures, is quite unnecessary. Under some circumstances certain drugs, however, can aid the treatment. I refer especially to the use of calomel at the beginning of the disease. Wunderlich thought he noticed that the administration of two to three calomel powders of 5 gr. (gm. 0.3) each in the first week of the disease had an ameliorating effect on its course, and sometimes even shortened it. This, of course, can scarcely be proved. However, I consider the exhibition of calomel useful, especially in those rather frequent instances in which there is constipation at the beginning of the disease. I do not employ the drug in the later stages, but when there are no other special indications, I prescribe only a simple muriatic-acid mixture. The question has been widely discussed as to the advisability of combating the fever through the administration of antipyretics.

The one-sided point of view which considered that the chief task of the physician in the treatment of acute febrile disease was to lower the temperature has been gradually abandoned. In describing the bath treatment of typhoid we have emphasized the fact that the action of the bath in reducing the temperature was only one factor, and perhaps neither the most efficient nor the most important factor, in the curative action aimed at. In giving antipyretics internally their antifebrile action is of chief importance, but some of them also have a certain sedative action on the nervous system. The important influence of the bath on the respiration and skin they do not possess. If we had to decide whether to treat typhoid exclusively by baths or exclusively by antipyrin and similar drugs, we should decidedly prefer the former. We would not wholly exclude the use of antipyretics internally in the treatment of typhoid, but we would limit their use more than is often the case. In our opinion they are indicated only when with existing high fever baths are for any reason impossible or contraindicated (*vide supra*), or when, in spite of the baths, the fever remains constantly high. In such cases the baths can often be judiciously combined with the use of antipyretics internally if the patient bears the drug well and feels better subjectively after the reduction of temperature than before. We often treat typhoid patients by baths in the day, and, if the fever be high, by a dose of antipyrin (15 to 30 grains, gm. 1 to 2)

at night. With severe headache, nervous restlessness, etc., this remedy is especially indicated; but we regard it as at least useless and often improper to give a patient with moderate fever large doses of an antipyretic without any sufficient reason, as unfortunately is often the case in practice; the only permanent result is to make the patient feel worse and upset his stomach. [In America, the use of antipyretics in typhoid and other acute infectious fevers is exceptional. They may occasion collapse, particularly if given just as a lysis or crisis is about to begin.]

In regard to the different antipyretics so frequently brought forward and recommended of late, we are of the opinion that antipyrin is most to be recommended. It was introduced by L. Knorr and first recommended by Filehne. In doses of 15 to 30 gr. (gm. 1 to 2), best given in wafers, it usually reduces the temperature considerably, although we may find that the height and the obstinacy of the fever are not identical terms. We do not often see unpleasant effects from antipyrin (such as vomiting, profuse sweating with defervescence, a chill when the temperature rises again, and sometimes a measles-like eruption). The patient often feels better than before while under the influence of antipyrin, since the drug, as we have said, also acts favorably on the nervous symptoms (headache, restlessness). The dose of 15 to 30 gr. (gm. 1 to 2) can in severe cases be repeated several times a day, but, as a rule, we should not exceed 80 or 90 gr. (gm. 5 to 6) in the twenty-four hours. One or two doses a day are usually all-sufficient. Lactophenin is also much recommended and even held as a specific in typhoid. In doses of 7 to 15 gr. (gm. 0.5 to 1) it reduces the temperature considerably and usually causes a marked improvement in the general condition. A total of 80 or 90 gr. (gm. 5 to 6) can be used in a day. Of the many other antipyretics which, of course, have often been tried in typhoid, we may first mention antifebrin (acetanilid), which in doses of 4 to 7 gr. (gm. 0.25 to 0.5) has a similar action to antipyrin (Cahn and Hepp), and it should be used in practice among the poor because it is much cheaper. If we avoid too large doses we seldom see unpleasant results. The appearance of a pale cyanotic hue of the skin is the only disturbing feature; this, as in anilin poisoning, is probably due to a change in the coloring matter of the blood, and therefore warns us to be cautious. Other new antipyretics are phenacetin (15 gr., gm. 1, at a dose), pyramidon (4 to 7.5 gr., gm. 0.25 to 0.5), salipyrin, etc., which have, however, no special advantages. Of the older antipyretics, quinin (in doses of 15 to 22.5 gr., gm. 1.0 to 1.5) is to be specially mentioned; indeed, it has recently been claimed that quinin has a favorable effect on the course of the disease.

Another important symptom which needs special treatment is intestinal hemorrhage. It has been already mentioned that if this occurs the baths should cease at once. Further than this, the chief remedies are ice and opium. Flat ice bags, cold-water coils, are laid upon the abdomen. The ice bags should not be too heavy, and should, if possible, be suspended from a hoop. Internally, the patient is given every two hours 15 or 20 drops of laudanum or a powder of 0.5 gr. or 1 gr. (gm. 0.03 to 0.05) of opium, either pure or combined with acetate of lead (opii, gr. ss., gm. 0.03; plumbi acetatis, gr. j, gm. 0.05; sacchari albi, gr. j, gm. 0.05). The object of the opium is to check peristalsis, and thus promote the formation of a clot in the bleeding vessel. [Morphin

subcutaneously (gr. $\frac{1}{8}$ to $\frac{1}{4}$) accomplishes the same purpose and more promptly. It should be repeated every half hour till beginning narcotism. If neither food nor drugs be given by the mouth for some hours, there will be more chance for the intestinal coils to keep quiet.] In severe cases we may try injections of ergotin, or fluid extract of hydrastis, 20 drops three or four times, or a subcutaneous infusion of about 7 ounces (200 c.c.) of a one- to two-per-cent sterilized solution of gelatin in seven-per-cent salt solution. For the threatening anæmia following severe hemorrhages, subcutaneous infusions of normal salt solution are to be employed. The baths cannot be resumed till there has been no bleeding for at least four or five days—and then only cautiously.

If peritonitis occurs, the treatment is much the same. Above all, opium must be used in still larger doses, but, unfortunately, as a rule, in vain. The surgical treatment of peritonitis has rather more of a future and more success, but experience of this is still scanty.

If there is considerable diarrhea, we can give mistura gummosa [P. G., gum arabic and sugar, each 15 parts; water, 170 parts] or small doses of opium, the latter sometimes combined with tannin, tannigen, etc. In general it is certainly proper not to check the ordinary moderate diarrhea of typhoid. Persistent constipation is always to be avoided. Constipation at the beginning of the disease is overcome by calomel (*vide supra*). In later stages we always try enemata first, to produce an operation. If this does not succeed, then we must employ rhubarb or castor oil. Great tympanites may be diminished by laying cold wet cloths or ice bags upon the belly. Considerable amounts of gas may often be removed by introducing a long rectal tube. As to puncturing the greatly inflated intestines, a method practiced by some physicians, we have no personal experience.

If there are severe pulmonary symptoms, baths or wet packs are, as we have said, the best remedies. Internally we may try liquor ammonii anisatus [P. G., olei anisi, 1 part; aquæ destillatæ, 24 parts; aquæ ammoniæ, 5 parts] and, especially with weak heart, benzoic acid (gr. ij to iij, gm. 0.1 to 0.2, in powder) and gr. iij, gm. 0.2 of camphor. If the pulse be very rapid, we may try an ice bag over the heart. If at the same time the pulse is small and weak, we give stimulants (camphor, strophanthus, caffein, and strong wine). We only rarely use digitalis in typhoid. If symptoms of severe cardiac weakness—so-called collapse—suddenly occur, a rapid and energetic interference is urgently demanded. Hypodermic stimulation should be employed and, above all other drugs, camphor in oil. Besides camphor, caffein injections (caffein nitrobenz., 2.0 in 10.0 = gr. xv in 5ijss. aquæ destillatæ, two to three syringefuls) may be employed, and wine, strophanthus, etc., internally. If the respiration stops it can sometimes be started up again by douches of cold water on the back of the neck. We may sometimes restore it by artificial respiration.

For nervous symptoms the baths and douching are the most effective remedies. The head is meanwhile covered by an ice bag. Antipyrin sometimes acts favorably. If there be great excitement, as shown by excessive restlessness or delirium, small doses of morphin internally, or, better, subcutaneously, are often very useful.

For insomnia, veronal in doses of 7.5 to 12 gr. (gm. 0.5 to 0.75), perhaps

in combination with 15 gr. (gm. 1) of sodium bromid, may be tried without hesitation.

The numerous other complications and sequelæ which may occur, but which cannot all be mentioned here, should be treated on general principles.

The prophylactic measures to avoid the spreading of the disease can be only briefly referred to. The most careful isolation of the patient is naturally of the first importance. If this be not possible in his home, he should be transferred to a hospital. Special attention should be paid to the very mild cases ("bacilli carriers," *vide supra*). The careful disinfection of the excreta (urine and feces) is of great importance. The best disinfectant is "Kresolwasser," as prescribed by the Royal Bureau of Health (2 ounces of liq. cresoli saponatus in a liter of water), which is added in equal parts to the feces, urine, or vomitus. Milk of lime, chlorid of lime, a two-per-cent solution of corrosive sublimate, lysol, etc., may also be employed.

We should further take care that bedpans, bedclothes, linen, the rectal thermometer, etc., should be handled by other persons as little as possible, and should be carefully disinfected.

The extensive investigations into the question of vaccination against typhoid have not led to any definite results, although they are by no means hopeless. [Late reports are encouraging.]

APPENDIX

PARATYPHOID FEVER

Schottmüller and others have repeatedly observed a typhoid-like disease, usually having a favorable course, in which the bacilli found in the blood, stools, and roseolæ have certain differential cultural characteristics which distinguish them from the typhoid bacilli. Thus far two types (Types A and B) have been identified. The blood serum of paratyphoid patients does not agglutinate typhoid bacilli, but does agglutinate the paratyphoid organisms. This circumstance is especially valuable in the diagnosis. The paratyphoid bacilli are, however, usually agglutinated by true typhoid serum. Further observations must be made to show the practical significance of the discovery of paratyphoid. It is worthy of mention that the so-called Bacillus paratyphosus B is often the cause of cases of "meat poisoning."

CHAPTER II

TYPHUS FEVER

(Spotted Fever. Petechial Typhoid. Febris exanthematica.)

Typhus fever is an acute infectious disease, perfectly distinct from typhoid fever, but formerly often confounded with it. The similarity of the two diseases, which led to their similar names, consists only in the grave general con-

dition with fever, and in a number of complications which may appear in both. There is, however, an essential difference in the whole course of the two diseases, and especially in the circumstance that the intestinal lesion which is characteristic of typhoid is never seen in typhus. The chief distinction between the two affections, which must undoubtedly lie in the difference in their causes, cannot yet be demonstrated. We do not yet know the organized pathogenic agents of typhus fever, although it must be presupposed that they exist.

Ætiology.—As to the way in which infection occurs, we have much less information even than in relation to typhoid. We know nothing as yet as to the specific agents which produce typhus, although microörganisms have been repeatedly found in the blood. It is an incontestable fact that the appearance of typhus in a place previously free from the disease is always to be referred to an importation of the pathogenic poison from without. It is likewise determined, through numerous observations, that typhus is one of the contagious diseases—that is, that the specific poison can be directly transferred from the patients to others around him. How it is transferred we have no certain knowledge. Perhaps the poison is contained in the expired air; or, as is still more probable, in the scales of epidermis; or, perhaps, in the other excretions and secretions of the patient. We are equally ignorant through what channel the infectious agent enters the system—whether it is inspired or swallowed. It is certain that the poison may be transferred in the clothes, etc., of the patient (fomites).

Favorable hygienic surroundings decidedly diminish the contagiousness of typhus fever. For example, in the well-ventilated pavilions of the Leipsic hospital there have rarely been cases of transfer of the disease to physicians, nurses, or other patients. On the other hand, if the hygienic influences be unfavorable, typhus fever may appear in very widespread epidemics. Those terrible epidemics which have been described under the names of "famine fever," "camp fever" (Hungertyphus, Kriegstyphus), etc., were for the most part typhus fever. In the smaller epidemics it is often possible to trace the disease to some wretched, overfilled tenement house.

At present typhus fever appears constantly in Great Britain. Ireland has been notorious for many years as a breeding place of the disease. It is also frequent in the eastern part of Germany (Posen, East Prussia and West Prussia, Silesia), in Poland, Galicia, Russia, and in parts of southern Europe. The isolated cases which occur every year here and there in central Germany, though more or less numerous, are, almost without exception, to be referred to an importation of the disease.

Typhus fever attacks by preference young adults of twenty to forty years; but it occurs in children, and is comparatively frequent in elderly persons. There is no marked dependence of the epidemics upon any particular season of the year. As in the case of typhoid fever, a person who has once had the disease seems to enjoy immunity from any fresh attack.

[The practical acquaintance of American physicians with typhus fever is, fortunately, limited. Many of the outbreaks which have occurred were traceable to immigrants, especially from Ireland.

During our Civil War the disease broke out neither among the armies in the field nor among the prisoners of war. A number of cases were reported at

the time, but great doubt has since been thrown upon the correctness of the diagnosis.]

Course and Symptoms of the Disease.—If we try to sketch the characteristic behavior of typhus fever, especially as contrasted with typhoid, we may say that the disease begins much more abruptly and rapidly, and that the fever quickly becomes very high and the general disturbance very severe, but the illness lasts a shorter time, seldom more than two weeks, and, with a favorable course, passes by crisis into recovery.

The duration of the period of incubation seems to vary. It is usually about twelve days—never less than four, or more than fourteen days. Sometimes, though not invariably, slight prodromata precede by some days the actual outbreak of the disease. These are languor, anorexia, headache, and pain in the limbs. Then the regular illness begins, as a rule, rather suddenly, and often with a pronounced rigor. With this the temperature rises quickly, and may on the very first evening reach 104° or 105° F. (40° to 40.5° C.). Vomiting is not rare, and may be repeated. A grave general condition, with fever, is developed in a few days. The patient feels exhausted. There is often violent pain in the loins and extremities. Nervous symptoms soon appear: persistent and intense headache, vertigo, spots before the eyes, ringing in the ears, and in many cases quickly increasing stupor and delirium. In severe cases the fever often reaches 106° F. (41° C.), and may be even higher, and it is almost constant, with but slight morning remissions. The skin is hot and dry, the tongue dry and thickly coated, the respiration moderate, the pulse very rapid. We very frequently find in the chest the signs of an extensive bronchitis. Nasal catarrh and conjunctivitis also occur. Serious intestinal symptoms are generally absent. The spleen is almost always greatly enlarged. The urine is concentrated and scanty, and sometimes has a trace of albumen. There is usually a moderate leucocytosis (in contrast to typhoid fever).

On the third to the seventh day of the disease the characteristic eruption appears. To this the disease owes its name of "spotted fever." The eruption consists of rose spots, generally very numerous and widespread, upon the trunk and extremities, often also on the face. Sometimes the spots are larger, and may then bear great resemblance to a fresh eruption of measles. The skin between the separate rose spots is not infrequently diffusely reddened. After two or three days the roseolæ become hemorrhagic, and change into lighter or darker petechiæ. It is commonly only in the lighter cases that the rose spots fade away without first becoming petechial. In rare though well-substantiated cases the eruption has been scanty, or even wholly wanting. Herpes does occur, but only seldom.

The fever begins to abate in light cases as early as the second week, coincidently with an improvement in the general symptoms. Often this change is indicated about the seventh day by a considerable remission in the temperature. On the other hand, in severe cases, all the symptoms grow worse. The weakness increases. The nervous derangement reaches the extreme of a severe "typhoidal state," expressed either by marked stupor, which sometimes passes into complete coma, or by violent delirium. Lobular pneumonia attacks the lungs, and the fever continues with unabated violence. These symptoms may end with death, but in favorable cases they decline rapidly. Sometimes this decline is preceded by a great rise in temperature (*perturbatio critica*), espe-

cially about the seventeenth day, rarely a few days earlier or later. In such cases the temperature is apt to fall by crisis, sinking in a day or two, with but slight interruption, down to the normal level. Even in those cases in which the descent is by gradations it is always decidedly more abrupt than in typhoid. The eruption quickly fades, the patients gradually improve, and, as a rule, become completely and permanently convalescent. It is true that some observers have seen relapses, but they are, at least in our present epidemics, extremely rare.

Complications and Varieties in the Course of the Disease.—From what we have said of its course, it is evident that the symptoms are essentially those of an intense general infection of the system. The sole demonstrable local lesion which is almost invariably present is the characteristic eruption, and this has evidently no causal relation to the severe symptoms of the disease. It is likewise extremely probable that most of the complications, which not infrequently arise in severe cases, are secondary, and occur in the way already described with considerable detail in the preceding chapter. They are just such complications as are possible in any severe constitutional disease, and embrace otitis, parotitis, extensive lobular pneumonia, more rarely gangrene of the lungs, and pleurisy; also furunculosis, purulent cellulitis, bedsores, dysentery, icterus, etc. Whether some of the local lesions which are observed may not be direct results of the pathogenic poison, we cannot at present decide. Among these would come, first of all, the rare cases of lobular pneumonia and nephritis. Sequelæ are, on the whole, rare, though sometimes there is a tedious anæmic condition, or neuralgia, paralysis, etc.

The separate epidemics of typhus present considerable variety, not only as regards the occurrence of individual complications, but more especially in the general course and character of the cases. For instance, some epidemics are distinguished by the greater frequency of light attacks (*typhus exanthematicus levissimus*). Here the entire attack runs its course in five to eight days. The fever is generally comparatively moderate; there are no severe general symptoms, and complications are exceptional.

Diagnosis.—It may be very difficult for a time to distinguish typhus from typhoid. The following factors are of chief importance: 1. The onset is much more abrupt in typhus than in typhoid, and is often accompanied by a pronounced rigor. 2. In typhus, the nervous disturbances usually appear earlier and are more severe than in typhoid. 3. The rash is seldom so extensive in typhoid as in typhus, and in typhoid it hardly ever becomes petechial. 4. In typhus the pains in the loins and limbs are generally much more pronounced. 5. The characteristic intestinal symptoms in typhoid. 6. The leucocytosis in typhus. 7. If we still find it hard to decide, the manner of recovery will almost always settle the question. Recovery in severe cases of typhoid is, on the average, much more tardy and gradual, by lysis. In typhus it occurs generally by the seventeenth day, and by crisis. Aside from these clinical differences, the surest means of excluding typhoid are the Widal reaction and the bacteriological examination of the blood (*vide supra*). It may, at the beginning, be difficult to distinguish typhus from smallpox, as well as from measles. The differentiation, however, is soon made by the general epidemiological considerations, and, above all, by the further course of the disease.

Prognosis.—The prognosis is chiefly determined by the severity of the fever and of the nervous symptoms. Extensive lobular pneumonia is the most frequent dangerous complication. The mortality varies greatly in the separate epidemics. It is sometimes only six or seven per cent, but may rise to twenty per cent.

Treatment.—Treatment is based on the same principles as in typhoid fever. There is no specific remedy. Besides good nursing, a judicious employment of baths is certainly our chief reliance for lessening the severity of many of the symptoms, such as febrile, nervous, and pulmonary disturbances, as well as for averting many dangerous complications. Potassium bromid (30 to 45 gr., gm. 2 to 3) is said to have a favorable effect on the delirious patients. Because of the marked contagiousness of the disease, typhus cases should, as soon as possible, be most carefully isolated. All the rules for disinfection should, of course, be carried out with the greatest care.

CHAPTER III

RELAPSING FEVER

(Relapsing Typhus—Febris recurrens)

Ætiology.—This disease was first named by English pathologists relapsing fever, and by Griesinger *febris recurrens*. It has a peculiar course, made up of separate attacks, and is further of great interest because it is one of the first infectious diseases in which the specific pathogenic organisms became known, and, being easily demonstrable in each separate case, were utilized for the speedy and certain diagnosis of the disease. Obermeier discovered in Berlin, in the year 1873, that in relapsing fever the blood, at certain times, invariably contains peculiar threadlike microörganisms. This discovery has since been universally confirmed; and it may be maintained that if once the presence of these "spirilli" be demonstrated in the blood, we are justified in making an absolute diagnosis of relapsing fever.

In Germany the disease did not become epidemic till the year 1868. In 1872 and 1873 there were considerable epidemics in Breslau and Berlin. Its last extensive appearance was in 1879 and 1880, when it spread over most of northern and central Germany, and was accurately studied by numerous observers. People of the poorer classes were almost exclusively attacked, and especially the "tramps." The uncleanly dens where these people lodge were found everywhere to be the chief centers of infection.

The transmission of the disease probably occurs mainly through bedbugs; they carry the spirilla-containing blood from the infected to healthy individuals.

The disease cannot be very contagious if the hygienic influences be good. At least the results of our late epidemics would imply this. In the Leipsic hospital, where at that time over two hundred and fifty cases were treated, and isolation could not be at all perfectly carried out, not one case of infection occurred. It is certain that the disease can be transmitted by direct inocu-

lation with the blood of patients. This has been established by a Russian physician, by the experimental inoculation of healthy persons. Doctors have been repeatedly inoculated at the autopsy of those who have died of relapsing fever. The disease may likewise be transferred by inoculation to monkeys, while other mammals seem to enjoy an immunity from it.

[The first cases of relapsing fever observed in this country were in Irish immigrants coming over in the same vessel in the year 1844. At several periods since then more or less limited outbreaks traceable to immigration have occurred, but the disease has never acquired any foothold with us, and comparatively few physicians have ever seen it. So far as can be learned, only one case has ever been seen in Boston, and that was in the person of a physician from another city, who brought the disease with him and passed through it in the Massachusetts General Hospital.]

Clinical History.—The stage of incubation lasts about five to eight days. It is only exceptionally that some slight prodromal symptoms present themselves just before the outbreak of the disease proper. As a rule, it begins suddenly, with a more or less pronounced chill and intense constitutional symptoms. There are violent headache, great languor, anorexia, and especially marked pains in the loins and extremities. The temperature rises rapidly, reaching generally 106° F. (41° C.) or higher as early as the first or second day. The skin is hot and dry, and usually quickly assumes a very characteristic dirty-yellowish color. In Leipsic we often saw herpes labialis, which seems, however, to have been rarer in epidemics elsewhere. The tongue becomes dry and thickly coated. Sometimes there is vomiting. The bowels are constipated, or there is a slight diarrhea. The spleen becomes rapidly enlarged, being, as a rule, even larger than in typhoid or typhus. The liver is slightly enlarged. The chest presents the signs of a bronchitis, generally moderate, but in exceptional instances severe. The pulse is much quickened. It is seldom that there are severe cerebral symptoms beyond a certain apathy and stupor. We have seen delirium tremens, sometimes, in drunkards. A very characteristic symptom, already mentioned, is the marked hyperæsthesia of the muscles, especially in the calves.

After these symptoms, accompanied by persistent and generally very high fever, have lasted five days to a week, there is a critical decline of temperature, with profuse sweating. The patient now improves so rapidly and decidedly that he thinks himself completely cured, and generally gives little credence to the physician's prophecy of a relapse. In rare but well-attested cases there has been really but one attack. The rule is that, after about a week, a second attack occurs, often a third after that, and, infrequently, even a fourth and fifth. In each of these, the above-mentioned symptoms are repeated more or less completely and violently. As the only certain and constant sign of the recurring attacks (the so-called relapses) is a fresh rise of temperature, it will be well to consider their peculiarities at the same time that we describe the course of the fever. During the intervals of normal temperature the other objective symptoms of disease are usually absent, except an evident splenic tumor, and, not infrequently, the peculiar pale-yellow hue.

Course of the Fever (see Fig. 5).—The beginning of the fever in the first attack is, as we have said, almost always sudden, so that it may even in a few hours reach a considerable height. The fever lasts, as a rule, five to seven

days, but not infrequently as short a time as three or four days, or as long as ten or twelve days. During this time it may keep a tolerably uniform height, but oftener there are considerable remissions, which may even come to deserve the name of pseudo-crises. In such cases the temperature sinks in the morning to normal or even lower, so that we might believe the fever ended;

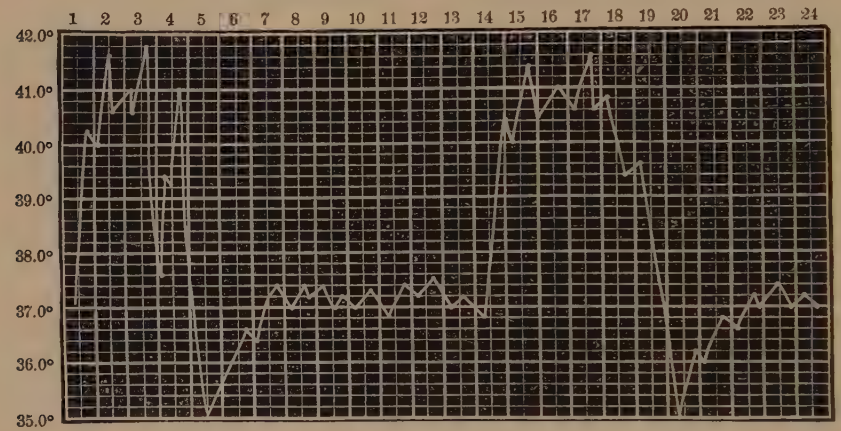


Fig. 5.—Example of the temperature curve in relapsing fever.

but in the evening the temperature rises again to its former level. These pseudo-crises are most frequent toward the end of the attack, but do occur sometimes in the very first days. The absolute height of the fever is, as a rule, very considerable. Temperatures between 105.5° and 106.5° F. (41° and 41.5° C.) are very often observed, and in themselves are not especially ominous in relapsing fever. The highest temperature we have observed was 107.9° F. (42.2° C.). Sometimes the temperature is more moderate (between 102° and 104° F., 39° and 40° C.). The fever almost always ends at the close of the attack by crisis, only rarely by a rapid, gradual decline. The crisis is often preceded by an especially great rise the evening before (*perturbatio critica*); so that the subsequent fall of temperature is very considerable. It generally occurs at night, and is accompanied by profuse perspiration. The fall may amount to 9° or 10° F. (5° to 6° C.). The temperature sinks almost always below normal, often as low as 95° F. (35° C.) or thereabouts. Once we saw it fall to 92.1° F. (33.4° C.).

To the first attack succeeds an interval during which there is no fever (apyrexia), which lasts on the average about a week, sometimes a less time, and often a greater. The longest interval we have ever observed lasted seventeen days. During this interval the temperature, which, as a rule, is at first subnormal, rises to normal, and then generally remains there. Exceptionally there are slight evening exacerbations to above 100.5° F. (38° C.). These may have no demonstrable cause, or may result from some complication, such as otitis, or a furuncle. Then comes another change, and generally a sudden one, ushered in with a chill, and a new rise of temperature, the beginning of the second attack or first relapse. During this attack the fever has the same general peculiarities as in the first attack. Generally the first relapse is briefer by a day or two than the first attack, but the reverse is sometimes true. We

will add that we have observed not infrequently a rather high evening temperature (101.5° F., 38.5° C.) for one or two days before the second attack began, as also before the third.

Relapsing fever seems in many epidemics to have been made up of two attacks, so that no more than one tenth of the cases had a third attack. On the other hand, the majority of the cases in the last epidemic had two relapses. In these cases the rule was for the interval between the second and third attacks to be one or two days longer than the first apyrexia; but earlier epidemics seem to have had the second apyrexia, if there was one at all, briefer than the first. The third attack is set down in all reported cases as decidedly shorter than either of its predecessors. It lasts generally two or three days. Exceptionally we have seen it persist for four or even six days.

A fourth and even a fifth attack may occur, but only exceptionally. If they do happen, they are usually imperfectly developed, and often are limited to a fever of one day's duration. The more accurately and persistently we take the temperature during convalescence, the oftener do we find slight rises of temperature occurring at intervals late in the history of the case. These are probably to be interpreted as final, rudimentary attacks.

The Spirilli.—The number of cases of relapsing fever in which no spirilli can be demonstrated in the blood, if the examination be accurate, has become so small that it can be disregarded, when we compare it with the much greater number of cases where such demonstration is made with ease and certainty. The best way is to get a drop of blood by pricking the skin, and examine it as it is, without mixing anything with it. Staining (with the basic anilin colors) is easy but unnecessary. With any good dry lens of 400 to 500 diameters the spirilli are seen with perfect distinctness. It requires a little practice to make them out, but this is easily obtained. Often the attention is first caught by little jogglings and motions of the red blood corpuscles, and then we see the delicate, narrow threads. Their length equals about three to six times the diameter of the red globules (Fig. 6). They exhibit an active and almost continuous motion, like snakes. Often the whole thread bends upon itself and then stretches out again. They are partly separate and partly tied up in knots composed of four to twenty individuals. The whole number visible in one field varies greatly in individual cases, and has no direct relation to the severity of the case. Often it requires long searching to find a few, while in other cases there may be twenty or more in the field at once. A very interesting fact is that their appearance in the blood depends upon the attacks of fever. On the first day of the attack we rarely find spirilli, and then only one or two. Upon succeeding days their number increases. Shortly before

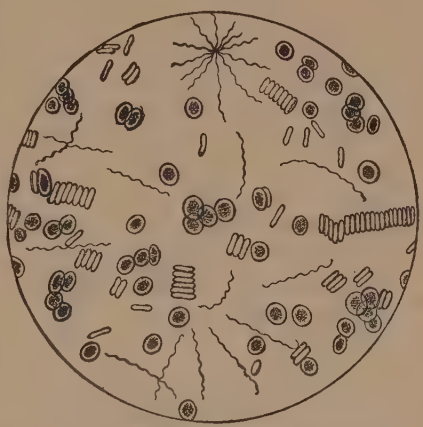


Fig. 6.—Spirilli of relapsing fever in the blood.

the end of the attack—that is, before the crisis—they generally disappear entirely; but even after the crisis they have been found, exceptionally and in very small numbers. They have very often been found by the author as well as other observers during the pseudo-crisis described above, so that, after the temperature has become normal, the presence of spirilli makes it very probable that another rise of temperature is impending. The spirilli have thus far been found in the blood only, in the catamenia, in bloody urine, or in blood coughed up from the lungs, and never in the organs or secretions (urine, milk, sweat, contents of herpetic vesicles). There can hardly be any doubt that the spirilli which appear in the separate attacks are to be regarded as separate generations. As to their manner and place of development we have as yet no knowledge. In the final, rudimentary attacks, we find few if any. If the patient dies during an attack, they are to be found in the blood after death. Artificial cultivations have not been very successful; nor have pure cultures of them, to our knowledge, ever succeeded. Albrecht states that they will subsequently develop in blood taken from a patient during the interval when he has no fever.

The blood is otherwise modified during relapsing fever. We very often find a slight increase in the white corpuscles. There is often a noticeable abundance of very small bodies, so-called granular elements (Körnchenbildungen). The significance of these (the remains of white corpuscles?) is still doubtful. There are, finally, peculiar cells, rather large, with fat granules. They were demonstrated by Ponfick in the venous blood, and are said to come from the spleen. We also find fatty-degenerated endothelium in the blood.

Complications.—Complications are, on the whole, rare, and mostly secondary. Important among these are troublesome ophthalmic disturbances, especially iritis and irido-choroiditis. Sometimes parotitis, laryngitis, or pneumonia occur. Epistaxis is a not infrequent complication. It is usually profuse and persistent, and it may even be dangerous. Sometimes there has been rather severe dysenteric trouble. In one case, which ended fatally, we observed a very peculiar intestinal lesion, consisting of hemorrhagic-necrotic foci in the colon and lower ileum. In severe cases acute hemorrhagic nephritis occurs with comparative frequency. At the autopsy an important and characteristic phenomenon are the wedge-shaped white spots which occur in the spleen, like infarctions. They have a clinical interest, as they may become the starting point of pyæmic conditions or of peritonitis. Splenic abscesses have been observed in a few cases.

Variations.—Variations in the course of the disease occur in this, as in all other acute infectious diseases. First, there are mild, abortive cases, in which the attacks are few and brief. Then cases have been described resembling intermittent fever. Of chief importance is that severe variety of relapsing fever first observed in Egypt by Griesinger, and described as "bilious typhoid." "Bilious typhoid" fever occurs in successive attacks, exactly like those of relapsing fever. The type is much more severe. As a rule, there appear marked icterus, grave nervous symptoms, hemorrhages into the skin and mucous membranes; and the termination is frequently fatal. The autopsy shows a greatly enlarged spleen, often containing infarctions and abscesses, and in some cases hepatic abscesses, septic nephritis, and similar lesions.

The two diseases have been heretofore considered either identical or closely

related—the association being based upon the alleged finding of the same spirilla in both cases, and on the alleged transmission of blood from a patient with "bilious typhoid" to a normal individual, with a resultant development in the latter of an ordinary recurrent fever! Recent studies cast considerable doubt upon these observations, so that the nosological situation of "bilious typhoid" is at present not decided upon.

Prognosis.—The prognosis of ordinary relapsing fever is on the whole very favorable. In the last epidemics the usual mortality was only two to four per cent. The fatal cases could some of them be laid to wretched nursing. In the remaining portion death resulted from complications, such as pneumonia and nephritis.

Treatment.—The treatment must as yet be purely symptomatic. Antipyretic treatment is generally needless, since the fever is relatively brief and often quite intermittent. Moreover, most patients cannot well endure cold baths, because the muscles are so painful. As a rule, good nursing and proper food amply suffice. If the muscular pains are very violent, we may order chloroform liniment as an embrocation. Complications are to be treated on general principles.

We are not acquainted with any remedy that can influence the disease itself or avert the relapses. Large doses of quinin, salicylic acid, etc., have been frequently employed for this purpose, but never with success. Lately there has been ascribed to calomel a favorable influence upon the general course of the disease, and its use is said to diminish the number of attacks. We must await further evidence in support of this statement.

CHAPTER IV

SCARLET FEVER

(Scarlatina)

We now begin the consideration of those acute infectious diseases which are usually embraced under the name of the "acute exanthemata." In this group are reckoned, besides scarlet fever, measles, German measles, smallpox, and varicella. Typhus may also be included in this group with a certain amount of justice. The point which these diseases have in common is that in all of them is developed a characteristic eruption, of slight clinical significance in itself, in most cases, but of thoroughly characteristic appearance in each disease, and hence of essential importance in diagnosis. A number of the acute exanthemata have this further point of mutual resemblance that they appear chiefly in children. These diseases are scarlet fever, measles, German measles, and varicella.

Ætiology.—Infection with the specific scarlatinal poison occurs almost always by contagion, which takes place very readily. A single approach to a patient ill with scarlet fever may suffice to communicate the disease. There is no doubt that the disease may be transferred by objects which the patient has touched, such as linen, clothing, furniture, or toys. Persons who have been

with the sick may be the means of transmitting the disease, although themselves unaffected. In England it has been thought that the contagium may be carried by milk. [Similar observations have been made in America (Kober, Taylor, Bell).]

Numerous observations show that the scarlatinal poison is with great difficulty destroyed, and that it may keep its contagious powers for months ("tenacity"). We can thus see how difficult, how impossible, it may be in an individual case to point out the source of contagion. Scarlet-fever patients can communicate the disease certainly as late as the end of the desquamative period, or perhaps somewhat longer.

Details as to the manner of contagion, or as to the specific poison itself, are as yet unknown. All statements made in regard to finding alleged specific microörganisms of scarlet fever are extremely doubtful. The exciting agent of scarlet fever must, however, be contained in the blood and in the contents of the miliary vesicles of scarlet-fever patients, for the disease has repeatedly been artificially produced in healthy persons by inoculation with these fluids.

Predisposition to scarlet fever is far less universal than is predisposition to measles or smallpox. In families with several children often only one or two fall sick, while the rest escape, although equally exposed. As age increases, liability to the disease greatly diminishes, although there are cases of scarlet fever among adults. The majority of patients are between two and ten years of age. Scarlet fever is rare during the first year of life. It is an interesting fact that children with fresh wounds, either accidental or surgical, are especially liable to scarlet fever. An analogous and familiar fact is that women after delivery have a strong tendency to the disease.[1] As a rule, a person is attacked but once, so that, after the disease is over, an immunity from contagion is enjoyed; but there are exceptions to this rule.

Scarlet fever is now spread over the entire globe. In Germany there are almost always some sporadic cases in the larger towns, while from time to time, especially in autumn, there are more or less extensive epidemics in one place or another. There is considerable variation in the different epidemics of scarlet fever, as in many other infectious diseases, in the general character of the disease, and above all in the prevailing mildness or severity of the cases and the frequency of certain complications (nephritis, diphtheria), etc.

Clinical History.—The period of incubation is not definitely known. Many observations seem certainly to indicate a short period of two to four days, but the incubation stage is perhaps often somewhat longer—four to seven days, and even more. There are hardly ever any decided prodromata. The disease begins rather suddenly, with fever, often introduced by chilliness, and sometimes by a well-marked rigor. There is almost invariably a painful, scarlatinal sore throat. A further symptom, in all cases of any severity, is cerebral disturbance, generally rather intense. There may be headache, dullness, uneasy sleep, delirium, and, in smaller children, sometimes even convulsions. A very frequent and characteristic early symptom is vomiting, which may be repeated.

The characteristic rash usually appears as soon as the close of the first day, or on the second, and begins on the neck and on the chest and face, quickly

[1] In puerperal cases genuine scarlet fever and septic diseases were formerly often confounded. (See Chapter XIX.)

becoming almost universal. The eruption consists at first of numberless small red points, crowded thickly together and soon united into a diffuse, intense, scarlet-colored erythema. The small and somewhat elevated points almost always correspond to the swollen hair follicles. The diffuse redness is the result of an excessive hyperæmia of the skin, and vanishes completely on pressure. The skin as a whole seems slightly swollen and thickened. The back usually presents the most vivid tint. In the face there is generally pallor of the lips and chin, presenting a very striking and characteristic contrast to the bright-red cheeks. If some object like the end of a penholder be drawn over the red skin, there soon arise corresponding white lines, due to contraction of the blood vessels. It is possible thus to make letters or pictures upon the back of the patient. We should add, however, that this is not a peculiarity of the scarlatinal eruption, being seen in other erythematous eruptions.

The rash persists for some three or four days, at first even increasing somewhat in vividness. It often appears more intense by artificial light than in the daytime. Meanwhile the severe general symptoms continue—the fever, the usually excessively rapid pulse, the cerebral symptoms, and the throat trouble. The spleen is often somewhat swollen, though seldom very large. Then the eruption begins to fade, the fever gradually ceases by lysis, the general condition and the difficulty in swallowing improve. With the end of the first week or the beginning of the second, the cases which run the typical course become fully convalescent. When the rash disappears, the epidermis usually begins to peel off, in a very characteristic way, in pieces of considerable size. The exfoliation upon the hands and feet is especially pronounced, and the little convalescents often amuse themselves by peeling off the epidermis in strips. Cases which are apparently the mildest and most benign may have their convalescence interrupted by the occurrence of a secondary scarlatinal nephritis. There is no sure prophylaxis against this.

We will now pass on from this general summary to a more complete consideration in detail of general and local symptoms; and we shall see how manifold are the clinical phenomena presented by scarlet fever.

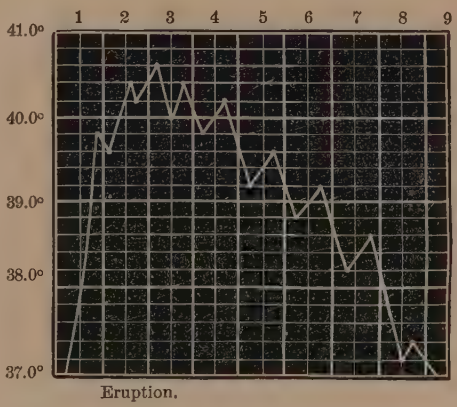


Fig. 7.—Example of a normal scarlet-fever curve.

1. *Fever* (see Fig. 7).—Although in a few undeveloped cases there is no fever, or scarcely any, almost all cases of any severity have high fever. It is only exceptionally that severe cases are observed in which the bodily temperature is little if at all elevated. As a rule, the fever rises rapidly upon the very first day, corresponding to the sudden onset of all the symptoms, to about 104° to 105° F. (40° to 40.5° C.). The next day it often becomes a little higher still, and then persists with but slight variations, as a rule, so long as the eruption it at its height. During this period a temperature of 105° F. or more (40.5° to 41° C.) is

not infrequently observed. When the eruption fades, and the other symptoms decline, defervescence occurs. This happens but rarely by crisis, and that in the slight attacks. It is almost always by prolonged lysis, as in typhoid, only more irregularly and more rapidly. If the fever lasts into the second week of the disease, it is almost always (though not without exceptions) caused by demonstrable complications. The most frequent causes are the persistence of a severe sore throat, the occurrence of inflammatory changes in the cervical glands, or a purulent otitis media. In closing what we have to say about the fever in this disease, we would emphasize the fact that the pulse is often very rapid (140 to 160 a minute), even in comparison with the height of the temperature.

2. *The Throat.*—The throat presents the most constant local lesion of scarlet fever. Sore throat is only in the rarest cases wholly absent;[1] but its form and intensity may vary extremely. The mildest variety is a simple, erythematous catarrh, without much swelling, but exhibiting usually a vivid and often punctate reddening of the soft palate and tonsils, the pharynx, and also the mucous membrane of the hard palate, frequently associated with enlargement of the little mucous follicles. Sometimes minute hemorrhages take place into the mucous membrane. In other cases the scarlatinal affection of the throat is from the start associated with considerable swelling of the parts, and especially of the tonsils, justifying the term "parenchymatous sore throat." Not infrequently small abscesses form in the lacunæ of the tonsils; or superficial spots of necrosis develop which leave behind them larger or smaller ulcers, and sometimes occasion considerable hemorrhage. There may even be a circumscribed gangrene of the tonsils.

The most important, because it is also the most dangerous, of the affections of the throat resulting from scarlet fever is the so-called scarlatinal diphtheria —that is, a "diphtheritic," or better, a necrotic inflammation of the tonsils and soft palate. This usually develops on the third, fourth, or fifth day of the disease, replacing a simple inflammatory condition of the parts. Whitish, dirty-colored spots develop on the tonsils, the arches of the palate, and the uvula. These rapidly increase in size, and cause a dry necrosis of the mucous membrane and subsequent ulceration. The process is a truly "diphtheritic" one—that is, there is an inflammation combined with an extension into the diseased tissues of a fibrinous exudation.

It is especially characteristic of scarlatinal diphtheria that there is almost invariably a considerable swelling of the cervical lymph-glands, except in those cases which die very quickly. It is true that the glands are usually somewhat enlarged in the milder forms of pharyngitis accompanying scarlet fever, but they seldom attain the size observed in the true diphtheritic process. In this there is an inflammatory and œdematous infiltration affecting often not only the glands themselves, but also the surrounding connective tissue, so that in severe cases the whole cervical region and the floor of the buccal cavity present a firm and usually a very painful enlargement. It should be added that the

[1] Absence of sore throat has been noted by others and by myself, especially in the scarlet fever of women in childbed. I suspect that the reason for this is that the infection does not enter in the usual way through the mouth, but from some wound due to parturition. On the other hand, it is to be noted that in the scarlet fever from wounds, when the infection follows an injury to the finger, etc., a sore throat occurs.

severity of the throat symptoms and the extent of the glandular swelling are not always commensurate. Almost always the scarlatinal diphtheria is associated with a marked stomatitis, and very often also with a severe purulent or even diphtheritic rhinitis, although an invasion of the larynx (*vide infra*) is only exceptionally seen. At the alæ of the nose and the corners of the mouth there are often superficial ulcers from this cause. Otitis is also a frequent complication of scarlatina (*vide infra*).

The influence of the scarlatinal diphtheria upon the general condition of the patient is always considerable. Apart from the marked local discomfort, there is often a severe general septic condition. Grave signs of cardiac weakness (a very rapid, small pulse) are very apt to appear early. Moreover, cases of scarlet fever associated with pharyngeal diphtheria often show at the same time other severe septic complications (inflammation of several joints, simple or purulent inflammation of the serous membranes, nephritis, etc.). Many cases end fatally in a few days, while others pursue a more tedious course, lasting perhaps several weeks before death comes. These are often associated with pyæmic processes in other parts of the body.

With regard to the pathogenesis of the throat troubles seen in scarlet fever, the more simple forms are in all probability directly associated with the scarlatinal process—that is, they are direct sequences of the affection. In regard, however, to the severer forms, and especially to the diphtheritic variety, it is almost certain that these are not a direct result of the scarlatinal poison, but are due to some secondary infection which occurs on the soil furnished by the primary scarlatinal angina. The severe necrotic disease of the mucous membranes and the secondary changes in the lymph-glands and other septic complications are due chiefly to streptococci (Löffler). Jochmann could also find the streptococci in the blood in almost all the severe cases. There seems to be a certain intimate relationship between the scarlatinal and the streptococcus infection; otherwise, it is very difficult to explain why such a characteristic secondary condition, the "scarlatinal diphtheria," develops only in scarlet fever. The latter and true diphtheria are, however, not identical. From a purely clinical standpoint they present several important points of difference. In particular, the scarlatinal diphtheria, in contrast with the primary form of the disease, seldom spreads to the larynx. The severe dyspnœa which sometimes develops in the course of scarlet fever is probably caused by an inflammatory œdema of the glottis. Paralysis of the soft palate, the ocular muscles, and other parts, is scarcely ever a sequel of scarlatinal diphtheria. Besides the streptococcus diphtheria, true diphtheria with all its sequelæ (laryngeal croup, etc.), sometimes complicates scarlet fever. It is identified by the presence of true diphtheria bacilli in the cultures.

3. *Parts Adjacent to the Throat.*—We proceed by a natural sequence to the consideration of the affections of certain parts adjacent to the throat, troubles which must be regarded as chiefly the result of direct extension, or of a conveyance of the inflammatory process from the throat.

The stomatitis we have already mentioned, as well as the disturbance in the neighboring lymph-glands and the surrounding tissue. Parotitis is not rare in severe cases. Of special importance is the scarlatinal inflammation of the middle ear, because it so often leads to permanent and serious disturbances of function. It not infrequently occurs in the desquamating period in the form

of a benign purulent otitis, but may, on the other hand, occur at the height of the disease in a very serious form, with a pronounced tendency to necrotic degeneration. This more serious form, as in the case of "scarlatinal diphtheria," is a streptococcus infection of a severe type. Sometimes, however, there is a true aural diphtheria. Destruction of the ossicles, the mucous membrane of the middle ear, and the tympanic membrane may occur in a few days. In especially severe cases the necrotic inflammation extends to the mastoid process, the labyrinth, the sigmoid sinus, etc., and may even lead to a purulent meningitis. It should be mentioned that this serious complication nearly always commences without any marked pain. It is therefore readily overlooked in the beginning, especially since the deafness of the little patients does not attract attention while they are so ill. When perforation of the drum membrane occurs, there is usually a purulent foul-smelling discharge. If the children recover from the scarlet fever, permanent difficulty in hearing very often remains. Statistics have shown that four or five per cent of all cases of deafness are referable to an attack of scarlet fever in childhood. Not a small percentage of cases of deaf-mutism is due to scarlet-fever otitis.

We have already spoken of the purulent or even diphtheritic rhinitis which almost always accompanies the scarlatinal sore throat. In rare cases there may also occur a purulent conjunctivitis, which is most probably the result of a direct conveyance of inflammatory secretions.

The tongue in scarlet fever deserves special mention. The first coating cleans off, and then the tongue usually presents a very characteristic appearance. It is diffusely reddened and covered with little elevations corresponding to swollen papillæ (strawberry or raspberry tongue, scarlatinal tongue).

4. *The Skin.*—The characteristic eruption, as developed in the great majority of cases, has been described above. It remains to describe certain variations from the usual appearances.

First, the eruption may be rudimentary. It is then not pronounced, and visible only on a limited portion of the body (face, trunk, or extremities).

Variations from the type are not rare; sometimes the papules are more strongly developed (*scarlatina papulosa*); very frequently there are little vesicles (*scarlatina miliaris*). This latter form of the eruption appears by preference upon the trunk, but it may come also upon the extremities, and is often brought out by excessive perspiration, or by wrapping up the patient too warmly. Many epidemics are noticeable from the frequent appearance of this miliary form. More rarely the rash has a spotted look, resembling the eruption of measles (*scarlatina variegata*). There may be minute ecchymoses, which are not very rare and not ominous. Well-developed cases of hemorrhagic scarlatina are, however, very dangerous, because here the general infection of the system is almost always exceedingly severe, and probably due to secondary sepsis; and there is besides, as a rule, a general hemorrhagic diathesis. Other cutaneous lesions, especially herpes and urticaria, are not so very unusual in connection with the scarlatinal eruption. Furunculosis has been repeatedly observed after the rash fades.

Desquamation generally begins as soon as the rash has completely disappeared, but it may not occur till a few days or even one or two weeks later. Its extent corresponds in general to the severity of the eruption, although extensive desquamation may follow a rudimentary eruption. It is seldom

bran-like or furfuraceous, as in measles. The rule is for it to be in lamellæ, so that, as we have stated, quite large strips of epidermis may be detached entire.

In rare cases an œdema of the skin appears after scarlet fever, which cannot be shown to depend upon nephritis (*vide infra*), but which may perhaps be due to an abnormal permeability of the walls of the cutaneous blood vessels following the eruption (*hydrops scarlatinosus sine nephritide*).

5. *The Kidneys.*—Next to the severer forms of throat trouble, the most important and dangerous complications are located in the kidneys. They may appear as early as the acme of the disease, as in many other infectious diseases. The urine has a trace of albumen. In rare cases the amount of albumen may be considerable. The appearance of the urine is generally not much changed, and the microscope reveals but few abnormal constituents. There are some white and red blood globules, a few hyaline casts, sometimes one or two renal epithelial cells. This initial albuminuria very rarely gives cause for alarm.

The genuine scarlatinal nephritis scarcely ever develops much before the end of the second or the beginning of the third week. Sometimes it comes even later. In one case under our own observation it did not begin till the thirty-third day of the disease. It may therefore be regarded to a certain degree as a localized relapse. It may be so mild as to cause no subjective symptoms whatever, so that it would be unnoticed if the urine were not carefully examined. On the other hand, it may be accompanied by the gravest symptoms, and may soon terminate fatally. It may follow either severe cases or the mildest, so that the rule should be to examine the urine in every case of convalescence from scarlet fever as often and as carefully as possible. No exact statement can be made as to the frequency of this complication, for it is much more common in some epidemics than in others.

The development of nephritis is often marked by a fresh rise of temperature. The elevation may be slight or it may reach 104° F. (40° C.). According to our own experience, the fever often comes a day or two earlier than the changes in the urine. As the nephritis goes on, it is very often accompanied by a moderate fever with remissions. This fever may be almost wholly absent, especially in mild cases. The pulse generally becomes harder, and is sometimes quickened; but in many cases it will be slow, and it is sometimes irregular. Among other objective symptoms, the first to excite notice is generally a slight puffiness of the face, which is usually pale. The eyelids, particularly, present an evident œdema. In the milder cases this œdema remains limited, while in others it gradually increases in extent and degree, involving first, as a rule, the dependent parts of the trunk, and later the extremities. Severe cases develop a pronounced anasarca. There are then, usually, effusions into the serous cavities, especially ascites and hydrothorax. The latter is dangerous chiefly through disturbance of the respiration, particularly if it be associated with severe bronchitis or pneumonia (*vide infra*). Although, in general, the development of a universal dropsy is characteristic of scarlatinal nephritis, there may sometimes be no œdema. On the other hand, it is noteworthy that the œdema may occasionally show a peculiar localization, especially in the mucous membranes (œdema of the conjunctivæ, œdema of the aryepiglottic folds with symptoms of laryngeal stenosis, œdema of the uvula and soft palate, etc.).

The urine exhibits the most important changes (*vide* the section on renal diseases). These may be insignificant in the milder cases, but they are very pronounced in the severe ones. The amount is much diminished. Sometimes there will be for several days almost complete anuria. In cases of any severity the urine is turbid, dark, often evidently bloody, with increased specific gravity (about 1.015 to 1.025), and containing a large amount of albumen. The sediment is generally abundant, and exhibits numerous hyaline casts of various lengths and diameters. To these may be attached red or white blood corpuscles, detritus, granules of hæmatoidin, or bacteria. In cases of some duration the casts are often moderately fatty. Very frequently there are found noticeably long and broad waxy [fibrinous] casts, which are opaque and yellow. In many cases of scarlatinal nephritis the urine is peculiar in having very many white blood corpuscles, either isolated or adhering to the casts. These undoubtedly originate for the most part in the kidneys. Red globules, some of them in the form of colorless rings, are found. They are usually present in small numbers, but may become more abundant, especially for a day at a time. We have seen a very hemorrhagic urine after the special albuminuria had disappeared. Renal epithelium is frequently seen, but not invariably nor in very large amount.

Uræmic symptoms often develop in scarlatinal nephritis. They may be of all degrees of severity. They will be described in detail under diseases of the kidney (*vide infra*). The uræmia may be so severe as to cause convulsions, coma, and death; but it is remarkable how often children recover from what seems to be the most pronounced uræmia.

The duration of scarlatinal nephritis varies greatly according to its severity. In cases which run a favorable course, the urine is generally abnormal for two to four weeks, or even longer. There is sometimes a very slight albuminuria present for months without causing any symptoms. Death may be due to uræmia or more frequently it may come on with severe dyspnœa. The latter is due partly to dropsical conditions (hydrothorax, ascites), or often to a severe diffuse bronchitis or pneumonia accompanying the nephritis. Cardiac insufficiency also may be the cause of death. The transition of acute scarlatinal nephritis into chronic nephritis, especially into chronic contracted kidney, is rare, but it has certainly been seen both by ourselves and others. It is worthy of note that, after the nephritis has lasted some four to six weeks, beginning hypertrophy of the left ventricle can be detected by the displacement and strengthening of the apex beat. Friedländer has demonstrated this early hypertrophy at autopsy.

The anatomical changes of scarlatinal nephritis can be mentioned only briefly here. The forms vary. We often find the "large white kidney"—that is, a diffuse acute nephritis with fatty degeneration of the epithelium—and usually also more or less numerous hemorrhages. In other cases the kidneys macroscopically are apparently little altered, but we see in the cortex the gray, prominent, bloodless glomeruli, in which the microscope shows very marked changes ("glomerulo-nephritis" of Klebs and others).

6. *The Joints.*—When desquamation begins, or even earlier, pain and swelling may attack a certain number of the joints. This trouble was formerly called scarlatinal rheumatism, but now it is usually known as scarlatinal synovitis. It is generally mild and quite temporary, but, exceptionally,

it may be severe and even purulent. Then it is usually a part of a secondary sepsis, as evinced by such other lesions as empyema, subcutaneous abscesses, jaundice, splenic tumor, nephritis, etc.; and they all seem to be caused by a secondary infection with streptococci, etc.

We have seen a few instances of excessive pain in the muscles of the thighs, accompanied by a moderate, diffuse swelling.

7. *Other Complications.*—Another important complication of scarlet fever is pneumonia. In severe cases lobular pneumonia sometimes appears as early as the first stage of the disease; but it occurs more frequently in connection with the nephritis, when it may have the significance of so-called nephritic pneumonia (*vide infra*). The respiration may be very seriously embarrassed by it. Inflammations of serous membranes in the chest—viz., endocarditis, pericarditis, and pleurisy—are more rare. They may or may not be accompanied by disturbances in the joints (*vide supra*). Pericarditis, endocarditis, and myocarditis sometimes develop during scarlet fever, but it is usually hard to decide whether they are due to the direct action of the scarlatinal poison itself or to secondary septic complications. We refer the tendency to tachycardia or bradycardia, irregularity of the heart beat, etc. (which sometimes lasts long after the scarlet fever), to myocarditic changes. Endocarditis may result in permanent valvular disease. Quite severe intestinal symptoms, such as diarrhea, may appear. These are generally the result of a catarrhal inflammation of the intestinal follicles. Dysentery is less frequent. The enlargement of the spleen has been already mentioned. The liver is sometimes found to be considerably enlarged. A general moderate swelling of the lymph-glands (neck, axillæ, groins) is usually present in scarlet fever at the time of the eruption. We usually find in the blood a pronounced leucocytosis which sometimes lasts long after the other morbid symptoms.

The marked increase in the eosinophile cells (up to fifteen to twenty per cent of all the white cells) is a very striking condition that is almost regularly present; it usually appears about the third day of the disease.

Variations in the Course of the Disease.—The diversities of the clinical picture in different cases of scarlatina will be understood when we consider the variety and number of the disturbances thus far cited. It is to be added that the general course of the disease may exhibit numerous peculiarities, of which it is hardly possible to give an exhaustive presentation. We will content ourselves with a cursory statement of the most important deviations from the typical course.

1. *Rudimentary Forms.*—To this class, in which the disease does not reach a perfect development, belong first the cases of simple sore throat with no eruption, or at most an extremely faint and partial one (*scarlatina sine exanthemate*). Sometimes even the sore throat is hardly to be seen, and there is nothing but a brief and slight fever with mild symptoms of general disturbance. The recognition of these cases as scarlatinal is possible only when we consider their ætiological relation to other undoubted cases of scarlet fever. We had an excellent opportunity to observe them when the disease broke out in the children's wards of the hospital at Leipsic. The diagnosis is sometimes confirmed by a slight though evident desquamation, affecting the hands, feet, legs, and back, or by an acute nephritis, which may follow the mildest attacks of this sort. Many cases of acute nephritis, though apparently

wholly spontaneous and primary, must be regarded as ætiologically scarlatinal.

2. *Rudimentary but Pernicious Forms.*—Under this head belong those attacks of scarlet fever in which the eruption is scanty or absent, while from the very start the most violent general symptoms appear. There is a very high fever, excessively rapid pulse, and delirium. Such cases must be the result of an uncommonly severe general infection. They usually end in speedy death. Other cases, ending fatally in a few days, have a well-developed rash without other localized disturbances.

3. *Severe Forms with a more Protracted Course.*—In these cases the long duration is not the exclusive result of especial complications, but is likewise due to the severity of the intoxication. One variety is the so-called typhoidal form of scarlatina, with persistent high fever and severe constitutional symptoms. Another variety is the hemorrhagic form briefly mentioned above, in which there are extensive hemorrhages into the skin and into the mucous and serous membranes. This form may run an extremely acute course. Further, in all pernicious forms, there may be severe local complications, particularly diphtheritic or gangrenous sore throat, inflammations of serous membranes, etc. Attacks of this sort are often not produced by the poison of scarlet fever alone, but by secondary complicating infections (*vide supra*).

4. *Relapses.*—In extremely rare cases relapses occur. After the first illness a fresh eruption breaks out with all the other symptoms of scarlet fever. In anomalous cases running a severe course, there is sometimes, at an advanced stage, a fresh, imperfect eruption (generally in spots), which Thomas has termed a pseudo-relapse. Probably this is usually a septic eruption.

Diagnosis.—The diagnosis of scarlet fever is made in most cases from the characteristic eruption taken in connection with the other symptoms. We should, however, bear in mind that exceptionally other eruptions appear which exhibit the closest resemblance to that of scarlet fever. 1. After the use of certain drugs, especially atropin (belladonna), quinin, antipyrin, morphin, chloral, etc.; more rarely after the ingestion of crabs, fish, etc.; and sometimes after the injection of antitoxic sera. 2. As a symptom of other infectious diseases, such as typhoid fever, smallpox; and, above all, in septic diseases (*vide infra*). In an uncertain or anomalous case, factors of importance for diagnosis are the ætiology (connection with other well-defined cases), the initial sore throat, and the eventual occurrence of desquamation or of a secondary nephritis.

Prognosis.—The prognosis must in every case be guarded. From what has been said of the course of the disease, it is evident that, even in cases which are at first apparently the most favorable, dangerous complications may appear. The commonest dangerous complication during the height of the disease is scarlatinal diphtheria, the commonest dangerous sequel is nephritis.

Treatment.—The majority of those cases of scarlet fever which take a typical course will recover completely without our aid. In these the task of the physician, so far as treatment is concerned, consists in arranging the details of hygiene and the general care of the patient. The sick-room should be cool and well ventilated and the diet rather strict, consisting mainly of milk. Broths and eggs may also be allowed. We should see that the skin and the mouth are kept clean. To change the linen frequently, if done with proper

precaution, is not only permissible, but very desirable. Rubbing the skin with olive oil, vaselin, etc., has some merit, and is especially to be recommended if the skin be harsh and dry after the eruption has faded.

[From the moment that the disease is declared, the patient should be thoroughly anointed daily with carbolized vaselin, lard, or the like; and this should be kept up until desquamation has ceased. Not only is the comfort of the patient promoted, but the danger of the spread of the infection is thereby greatly lessened.]

The scarlatinal disease of the throat must be treated with the greatest attention, the main duty of the physician in this regard being to prevent, if possible, the ingress of the above-mentioned secondary infection. It is therefore our opinion that in every case of scarlet fever the greatest pains should be taken from the very commencement of the disease to maintain complete disinfection of the mouth and throat. Larger children may use a gargle of chlorate of potash, hydrogen peroxid, borax, permanganate of potash, sage tea, etc. Inhalations of hydrogen peroxid, boric acid, etc., are also to be recommended where practicable. If there is prostration, or if the child be young or willful, we may frequently cleanse the mouth and throat by means of a spray apparatus. Sometimes it is a good plan to let the patient swallow slowly a half teaspoonful of a solution of potassium chlorate (about 1 to 40), every half hour or oftener, with the object of contributing to the local disinfection of the throat. If scarlatinal diphtheria nevertheless develops and the cervical lymph-glands begin to increase further in size, there is reason to hope, according to the experience of Taube and Heubner, that parenchymatous injections into the tissue of the tonsils or the palatine arches may yet check the spread of the secondary infection. About 6 minims (a Pravaz syringe half full) of a three-per-cent solution of carbolic acid may be injected twice daily upon each side by means of a long hollow needle and a subcutaneous syringe. Catti recommends painting the affected parts with a 1 to 1,000 solution of corrosive sublimate. Of the many other remedies advised, we may add that the insufflation or dusting of the tonsils with powdered sugar is of advantage. In ordinary scarlatinal diphtheria we can expect no benefit from Behring's serum therapy, but if there is a complication with true diphtheria a serum injection is most advisable.

Numerous attempts have recently been made by Moser and others to treat the serious secondary streptococcus infections with an antistreptococcus serum (" scarlet-fever serum of the Höchster Farbwerke "). Although good observers (Escherich) have reported favorable results, a definite conclusion is not at the present time possible. At any rate, it is justifiable to try the serum treatment in severe cases.

If the nose be likewise affected, the chief thing to do is frequent cleansing and syringing while the head is bent forward. We should be on the watch for the possible occurrence of otitis. In this particular the physician is often guilty of sins of omission. We would particularly emphasize the importance of careful watching of the drum membrane and the mastoid process in every case of scarlet fever, because timely interference (paracentesis of the drum membrane, etc.) may at least prevent a great deal of mischief. The treatment of the purulent ear discharge (irrigations, etc.) must be carried out according to general otological principles.

Inflammation of the glands in the neck, if severe, is prone to pass on to suppuration, and it must then be treated surgically. Ice is generally not so well borne as warm applications (poultices or warm bran cushions).

If there be continuous high fever, accompanied by rather severe constitutional symptoms, a moderate employment of the cold-water treatment in scarlet fever is to be strongly recommended. The baths seldom need to be cooler than 81° to 88° F. (27° to 30° C.), and are to be employed two or three times daily. If the nervous disturbance be serious, or if the respiration be impaired, the patient should be douched with cold water during the bath. Instead of baths, wet packs may be used to advantage. Internal antipyretics, such as antipyrin, may usually be dispensed with, although in private practice we may be obliged to employ them.

We must watch the condition of the heart carefully. When the pulse is very rapid we may use an ice bag on the heart. When the heart begins to grow weak we may prescribe caffein, tincture of strophanthus, wine, etc., and the most efficient remedy for threatening collapse is injections of camphor. With mild scarlatinal arthritis salicylate of sodium and antipyrin act well as palliatives, but in severe cases they, of course, do little good.

We know of no means to avert scarlatinal nephritis. In justice to himself, the physician must always at the start point out the possibility of its occurrence, and must avoid as far as possible errors in diet or exposure to cold on the part of his patient. He may thus escape blame. The treatment of the nephritis and its results are fully described in the section on renal diseases (Vol. II). The most important remedies are a suitable (milk) diet and the use of warm or possibly hot baths and packs.

The patient must, as a rule, keep his bed three or four weeks, even if convalescence be uninterrupted.

[This injunction is rather extreme. Nephritis is as likely to follow a mild as a severe case, and occurs sometimes in spite of every precaution. The physician should use his discretion as to the length of time the patient is kept in bed, carefully guarding against exposure to cold and imprudence in diet.]

After desquamation is completed the skin should be thoroughly cleansed by warm baths. The disease is so dangerous that, whenever a case occurs in a family, isolation is absolutely demanded, and, if possible, all the other children should be sent away. If this advice be disregarded, we can reject all responsibility for any further cases and their results.

According to the laws almost universal in Germany at the present time, brothers and sisters of the patient may not attend school for at least six weeks. Everything that has come in contact with the patient (linens, clothes, toys) should either be destroyed or thoroughly fumigated. The sick-room should be cleansed, aired, and disinfected at the termination of the disease.

[Scarlet fever is a disease at once so highly contagious and so common that it may be taken as the type of its class. Its hygienic treatment and the measures needful to prevent its spread consequently deserve more minute detail.

The sick-room should be at the top of the house, if possible, and exposed to the south; every unnecessary article of furniture and all ornaments should

be removed beforehand, carpets, curtains, and stuffed or upholstered furniture being included. A window should be kept open constantly, top and bottom; in cool weather a fire should be burning; in warm weather ventilation is furthered by placing a gas burner or large kerosene lamp near the throat of the chimney. Outside the door of the sick-room a sheet moistened with a disinfectant solution should be carefully hung. Only those whose presence is absolutely necessary are to be allowed in the sick-room, and the physician, when his visit is completed, should pass directly out of the house.

A convalescent should be kept away from all who are liable to contract or convey the disease until desquamation has entirely ceased. Several warm soap baths should be given before the child emerges into everyday life, and it should finally be dressed in uncontaminated clothing.]

CHAPTER V

MEASLES

(Morbilli)

Ætiology.—In contrast with the malignancy of scarlet fever is the comparatively benign nature of measles, a disease of childhood which is but little feared even by mothers. It is so widespread, and the susceptibility to it is so universal, that measles passes for an almost unavoidable but comparatively insignificant annoyance. Indeed, few escape it; and probably the reason why adults have measles so much less frequently than children is simply that most adults have already suffered from it in childhood. A second attack of measles in the same individual may occur, but it is certainly rare.

[In highly civilized countries measles has prevailed so long that it would seem that a relative resistance against the poison has been acquired. The frightful ravages of the disease when it was planted in virgin soil, as among the Fiji Islanders not many years ago, apparently bear out this view.]

Measles generally comes in epidemics. Sporadic cases are exceptional. In this respect measles differs decidedly from scarlet fever. The rapid spread of the disease when it has once broken out is a result of its great contagiousness. If one child in a family is attacked, the others almost always take the disease. The infection may be transmitted even by well persons and by means of articles with which the sick have come in contact, although this is not often the case. We are not yet acquainted with the specific poison of measles—although its existence is to be taken for granted—nor with the details of its transmission. Still, it seems most probable that the poison is inhaled through the mouth and nose, and that this is the reason why its effects are usually first developed in the respiratory passages (*vide infra*). The disease can be artificially produced by the inoculation of healthy children with the blood or liquid secretions of those suffering from it.

Clinical History.—The length of the stage of incubation is tolerably uniform. It is ten days to the beginning of the first symptoms, and thirteen or fourteen days to the breaking out of the eruption. These figures have been

established by the observations of Panum, the opportunity having been afforded upon the first introduction of the disease into the Faroe Islands. As a rule, there are no especial prodromata during the period of incubation except some slight elevations of temperature. At the end of ten days the initial stage [1] begins, generally suddenly, and with an abrupt rise of temperature to 102° or 104° F. (39° to 40° C.). At the same time the characteristic catarrhal symptoms appear: nasal catarrh (coryza), to be recognized by the abundant nasal secretion, the frequent sneezing, and sometimes also by nosebleed; more or less severe conjunctivitis, characterized by photophobia, reddening of the eyes, and increased flow of tears, and, lastly, symptoms of a catarrh of the upper part of the respiratory tract, usually moderate, but nevertheless causing hoarseness and a slight cough. With all this the general condition is disturbed, the children are restless, have headache, and eat little. Symptoms of a mild sore throat are not infrequent, but are very far from being so prominent as in scarlet fever.

The smaller children are greatly annoyed by mucus collecting in the nose or nasopharynx. In most cases, on the second or third day of the disease, the characteristic initial mucous membrane exanthem appears. It occurs on the mucous membrane of the soft and hard palate in the form of irregular red macules, the size of a lentil or even larger. About the same time, or even earlier, punctate, white, scale-like spots, often surrounded by a red areola, appear on the buccal mucous membrane opposite the molar teeth (the so-called Koplik's spots).

These initial symptoms last, as we have said, three or four days. Then the eruption begins (stage of eruption). The true eruption of measles begins almost always in the face, on the cheeks, forehead, and around the mouth (contrasting with the characteristic pallor of the chin in scarlet fever), and spreads from there rapidly downward over all the rest of the body. The eruption consists at first of little papillæ, corresponding to the follicles. These are soon surrounded by a pale-red, slightly elevated border, and in many cases become confluent. Perfectly flat elevations, of various sizes and of extremely irregular, dentated, roundish, or angular shape, develop. They are often so thickly crowded together as to touch one another, but usually limited portions of normal skin intervene between them. Within each raised spot the little follicular papillæ remain visible.

With the beginning of the eruption the fever rises, having been, as a rule, slight during the last days of the initial stage. It attains about 104° or 105° F. (40° to 40.5° C.). In thirty-six to forty-eight hours the eruption reaches its full development and its greatest extent. The fever and the catarrhal symptoms also persist for the same length of time. Sometimes we find a slight swelling of all the lymph-glands. Then follows a decline in the fever, usually rapid, and indeed almost by crisis, while the eruption after a short period of full development begins gradually to fade during the two or three days following. At the same time the catarrhal symptoms diminish. A more or less extensive desquamation of the epidermis begins, scarcely ever in large pieces, as

[1] We consider the term "initial stage" more correct than "prodromal stage." The "prodromal symptoms" are the first slight symptoms which occur during the time of incubation of an infectious disease, while the symptoms presented by measles before the breaking out of the eruption are a part of the already developed disease.

in scarlet fever, but in little scales like bran. In eight or ten days, if the disease runs a normal course, the patient is fully convalescent.

After this brief description of the usual course, we must consider more closely some of the symptoms and possible complications.

The fever (see Fig. 8) of measles exhibits, as has been already implied, a tolerably typical course. It begins with a rather marked and rapid rise upon the commencement of the disease. On the morning of the second day there is usually a marked remission, often to normal. In the last two days of the initial stage the fever is moderate, very rarely being so high as at the beginning. With the eruption there is a new, rapid rise, usually higher than the initial one, so that we may well divide the fever into two periods—the prodromal fever and the eruptive fever. This latter is but brief and does not persist, as in scarlet fever, during the entire duration of the eruption. It falls by crisis when the rash has attained full development. There may, to be sure, be slight elevations of temperature during the next day or two; but, if the fever is considerable and persistent, it is always a sign that complications have arisen, probably in the respiratory apparatus.

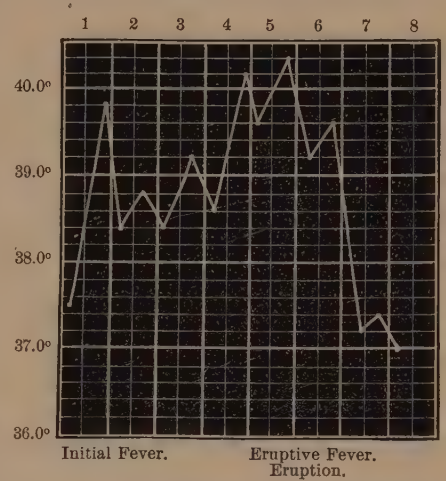


Fig. 8.—Example of the temperature curve in measles.

The eruption usually assumes the form described above, but may present manifold varieties. Sometimes its development is rudimentary. Sometimes it does not begin on the face, but on some other part of the body. This is generally regarded as a sign that the case will be anomalous in other ways as well. The individual spots may be smaller than usual, and may remain entirely separate from each other (*morbilli papulosi*). In other cases the eruption is so confluent (*morbilli confluentes*) that it resembles the eruption of scarlatina. The formation of vesicles (*morbilli vesiculosi*) also occurs, but much more rarely than in scarlet fever. Hemorrhagic measles is also observed, but usually only in the form of small, capillary bleeding, and in cases that otherwise run a perfectly favorable course. Very rare cases have indeed been described, with a general hemorrhagic diathesis and severe symptoms, resembling hemorrhagic scarlatina (so-called "black measles"). In addition to the proper eruption of measles, other eruptions sometimes develop—such as vesicles, wheals, and pustules.

The pulse is generally not so rapid in measles as in scarlet fever. Enlargement of the spleen can be made out only to a slight degree, if at all. As a rule, we find no pronounced leucocytosis in the blood. Indeed, in striking contrast to scarlet fever (*q. v.*), there is usually a pronounced leucopenia at the height of the disease. It is only in the period of incubation and with the advent of complications (pneumonia, etc.) that leucocytosis is present.

Complications.—The complications of measles are for the most part exaggerations or abnormal varieties and extensions of those troubles which are observed during the usual mild course of the fever. As in scarlet fever (*vide supra*), we often have to deal with the effects not of the original, but of secondary infections. Compared with the great majority of mild attacks taking the typical course, cases presenting complications of any severity are rare, and much less frequent than in scarlet fever. Epidemics are only now and then distinguished by unusual severity.

Often quite serious eye diseases are developed, particularly blennorrhagic conjunctivitis, keratitis, and iritis.

Marked inflammation of the mucous membrane of the nose, throat, and larynx may prolong the course of the disease. This is often merely an exaggeration of the usual catarrh. Otitis media likewise sometimes occurs. A laryngitis of marked intensity, with considerable swelling of the parts involved, may produce much discomfort, or even symptoms of stenosis ("false croup"). Actual croupous and diphtheritic lesions of the throat and larynx also occur (diphtheria of measles). This last is indeed much rarer than scarlatinal diphtheria, but it may have the same unhappy termination. It is worth mentioning that sometimes genuine laryngeal croup is observed in measles, unaccompanied by lesions of the pharynx.

It is, however, in the lungs that the most frequent and important of all complications in measles occur. The usual mild bronchitis becomes very intense, extends into the bronchioles (capillary bronchitis), and then results, for the most part, in a more or less extensive, lobular, catarrhal pneumonia (*q. v.*). According to Köster, measles pneumonia rises from an inflammatory proliferation of nuclei about the small bronchi, and it usually invades the neighboring alveoli by continuity along the small branches of the pulmonary artery. It is then a "small nodular," but not strictly a lobular pneumonia. Measles pneumonia is to be diagnosticated when moist râles are heard in abundance over a large part of the chest, and when there is at the same time persistent fever and pronounced difficulty in respiration, with cough, dyspnœa, or cyanosis. We get decided dullness on percussion only when the separate centers of infiltration are more than usually confluent. Genuine lobar, croupous pneumonia appears much less often than the lobular variety. It attacks one lobe, or several, is attended by high fever, and may end with a well-marked crisis.

The foregoing pulmonary symptoms usually appear at the height of the disease, and persist after the eruption fades. They may delay convalescence for weeks. In other cases measles will be seen at the start to run a normal course, temperature will have already fallen, and then come new fever and the appearance of decided pulmonary disturbance. This is always to be regarded as a grave complication; and especially in feeble children it may lead to death, with the symptoms of impaired respiration or of constitutional exhaustion. At present we cannot definitely state how far in measles pneumonia we have to do with the direct action of the measles poison itself or with secondary infection (streptococci, diplococci, etc.).

Marked intestinal symptoms sometimes appear, particularly an excessive diarrhea, due to intestinal catarrh. It is characteristic of measles that in severe cases such a diarrhea may assume a pronounced dysenteric character,

indicated by blood and slime in the dejections, symptoms which usually depend upon the development of follicular colitis with ulcerations.

Now and then still other complications may present themselves, of which a full enumeration is impossible. Nephritis does occur, but far less often than in scarlet fever. A simple albuminuria during the acme of the disease is not infrequent, but, as a rule, it has no especial clinical significance. We should mention gangrene of the cheek, the so-called noma, as a complication, which is very rare but apparently characteristic.

Peculiarities in the Course of the Disease.—Peculiarities in the course of the disease are much rarer in measles than in scarlet fever. Yet we see, on the one hand, unusually mild or rudimentary cases, in which either the rash or the other local symptoms are remarkably slight, and, on the other hand, abnormally severe cases. These latter are distinguished by the unusual height or persistence of the fever, by the severe constitutional and nervous symptoms, and further by the early appearance of complications. Such cases have been termed "typhoid measles." We have already mentioned the severe form of hemorrhagic measles.

We should notice the clinical relation which measles bears to two other infectious diseases—to whooping cough and tuberculosis. Measles and pertussis (*q. v.*) may follow each other in the same individual at a short interval, sometimes one and sometimes the other taking the initiative; epidemics of the two diseases prevail with comparative frequency at the same time. Tuberculosis is likewise to be mentioned as an important sequela of measles. In these cases it is probable that the children have a tuberculous focus smoldering somewhere in the body (lymph-glands, lungs) before the attack of measles which kindles it and aids its further extension. The measles may also, of course, excite a predisposition to infection with tubercle bacilli.

Diagnosis.—The diagnosis of measles, as of the other acute exanthematous diseases, is based chiefly upon the eruption, but we must also regard, of course, other symptoms which may be present (fever, catarrhal symptoms). Personal experience does more to sharpen the perception than can the fullest descriptions. We can merely suspect the disease during the initial stage unless an epidemic prevails. If, besides the characteristic catarrhal symptoms, the above-mentioned eruption on the mucous membrane of the palate exists, or if Koplik's spots are present on the buccal mucous membrane, the diagnosis becomes tolerably certain. We should consider that eruptions similar to that of measles appear in other diseases, more especially in German measles, scarlet fever, typhus fever, in the beginning of smallpox, and in secondary syphilis (roseola). Furthermore, we need to exclude eruptions which are due to drugs, especially antipyrin, and also turpentine, balsam of copaiba, etc. In cases which are doubtful we shall be enabled to form a decided opinion by a consideration of the other symptoms, and, above all, by the further course of the disease.

The difference in the blood picture in scarlet fever and measles is of value in their differential diagnosis—leucocytosis and eosinophilia in scarlet fever, leucopenia in measles.

Prognosis.—We have already remarked how favorable in general the prognosis is, but we must here repeat that all epidemics do not exhibit the same benign character, and that in every case the physician must bear in mind the

possibility of complications, and particularly the danger of severe pulmonary disturbances.

It should be mentioned that measles frequently seems to run a severer course in adults than in children.

Treatment.—The patient should in general be kept somewhat warmer than in scarlet fever. Even in what seem to be the mildest cases the child should be kept in bed till desquamation is over. The sick-chamber is to be somewhat darkened, on account of the photophobia which usually exists at first. In this way, normal cases run on favorably without any especial therapeutic interposition. The catarrhal symptoms, however, should always be heeded, since to disregard them may lead to their becoming aggravated. The chief requisite is cleanliness. At regular intervals the eyes, the nasal cavity, and the mouth should be washed out and douched with lukewarm water.

If, despite all this, certain disturbances appear in a worse form than usual, or if complications develop, these must receive especial attention. Severe eye troubles should be treated according to the usual ophthalmological practice. The treatment of croupous trouble in the throat or larynx will be fully described in a later chapter. For the pulmonary complications, lukewarm baths, combined if need be with rather cool douches, constitute the most effectual remedy. Cold packs are also very efficient. These we shall speak of later in more detail in the chapter on catarrhal pneumonia. The chief task of the physician in the treatment of measles is, however, to hinder as far as possible the appearance of complications. We are not acquainted with any internal remedies for the lung troubles which are at all trustworthy. In rare instances the excessive accumulation of mucus in the bronchi requires the administration of an emetic. As expectorants we may try ipecac, liquor ammonii anisatus, etc. If considerable intestinal disturbance arises, we must employ small doses of opium, calomel, tannalbin, or subnitrate of bismuth. We hardly need to say that, whatever else is done, the strength of the patient should be kept up as much as possible by giving wine, broths, milk, eggs, etc. For at least two or three weeks after the disease has ended, the child must be very carefully watched.

As the disease is usually so mild, prophylaxis is not very strenuously attempted. If one child in a family is attacked, it is probably already too late to isolate the others, and it is even an advantage to the family to have all the children finish at once what they will hardly be able eventually to avoid. We would make an exception in favor of isolation if the disease prevailed in a severe form.

[It is not customary with us to insist so strongly upon isolation and thorough disinfection as in scarlet fever, but the tendency of the present day is toward a wide application of the principles of preventive medicine. It is certainly of no advantage to a child to contract measles. Delicate children, especially those with tuberculous predisposition, should be carefully guarded against it; and, even if it is decided that it is not worth while to attempt to confine the disease to one member of a family, every precaution should be taken against infecting other families. Under suspicious circumstances, consequently, children are to be kept away from school and from contact with others.

If there is any reason to fear the development of tuberculosis, every possible hygienic means should be employed in order that full vigor may be regained.]

CHAPTER VI

RÖTHELN

(German Measles. Rubella)

Röthelн is a disease similar to measles, but distinct from it, although formerly often confounded with it, and perhaps with scarlet fever as well. The observations of Steiner, Thomas, and others leave now no room to doubt that these diseases are distinct, for epidemics occur in which all cases present the characteristic peculiarities ascribed to rötheln. But the best proof is that children who have had rötheln are not infrequently attacked by genuine measles later. It may indeed be very difficult in an individual case to decide which disease is present; but that rötheln does exist, as an independent form of disease, can be denied by those alone who have never seen it.

After an incubation of about two or three weeks the disease begins with the appearance of the eruption, which is usually noticed first in the face. Initial symptoms (cough, sneezing, etc.), preceding the eruption, either are wholly absent or at most last for half a day. The eruption is decidedly like that of measles, but its individual spots are smaller. They are seldom larger than small peas and circular, being only exceptionally as dentated and irregular in outline as are the maculæ of measles. They appear on the whole face, the head, the trunk, and the extremities, are pale red (sometimes deep red), only slightly elevated, and are not apt to become confluent. In rare instances, small vesicles develop upon the macules. The soft palate sometimes exhibits, as in measles, a faint macular eruption at the beginning of the disease. After three to four days the eruption fades. There is usually no decided desquamation.

Other symptoms of disease than this eruption are slight. Fever in many cases appears to be entirely absent. As a rule, there is for a day or two a little elevation of temperature, reaching 102° F. (39° C.) at most. Tokens of a moderate catarrh of the conjunctiva, the nasal mucous membrane, the throat, and the larynx are also observed—viz., photophobia, nasal discharge, and cough. Often, the cervical lymph-glands are more or less swollen. The constitutional disturbance is generally so slight that the child can hardly be kept in bed. Important complications hardly ever occur.

The prognosis of rötheln is therefore perfectly favorable, and the employment of any special treatment is needless.

CHAPTER VII

SMALLPOX

(Variola. Varioloid)

Ætiology.—Smallpox has been known for centuries, although formerly often confounded with other diseases.[1] It is one of the most dreaded acute

[1] The very name smallpox (*petite vérole*) is significant of its confusion with syphilis, which was termed the "great pox."

infectious diseases, and in earlier times it has destroyed thousands in its pestilential progress. It was the discovery of the possibility of prophylactic inoculation, and the ever-increasing spread of this precautionary measure, which first robbed the disease of some portion of its terrors.

Despite numerous studies (L. Pfeiffer, Guarnieri, Wasilewski, and others), the specific agent of smallpox is not known. The so-called Guarnieri vaccine bodies—small, round bodies found in the cells of smallpox and vaccine pustules, that stain deeply and have nucleus-like centers—do not appear to be protozoa, but rather extruded nuclei. Their presence is, however, so characteristic of smallpox that they may serve in the differentiation of that disease from other pustular affections, more especially varicella (*vide infra*). The Guarnieri bodies are most easily found in the characteristic pustules that appear on the rabbit's cornea after the latter has been inoculated with the contents of a smallpox pustule (Jürgens).

[Councilman and his colaborers have described the whole life history of a protozoön in the skin lesions, with confirmation by Calkins and by Howard and Perkins.]

Predisposition to variola, except as diminished by vaccination (*vide infra*), is universal. The disease may appear at any age, even *in utero*. Women are believed to be especially liable to it during pregnancy and child-bed. It is said that persons ill with another acute infectious disease, such as scarlet fever, measles, or typhoid fever, are, for the time being, tolerably secure from infection with smallpox; but this rule certainly has exceptions. The explanation of the fact that the most widespread epidemics of smallpox generally occur in winter is to be found in the closer living quarters of humanity in the cold season. The same individual rarely takes the disease a second time.

A case of variola is always the result of transmission of the poison to a healthy person from one who is already ill with it. The specific poison is most abundant in the diseased portions of the body and in the pus of the suppurating pocks, as well as the crusts and scales which are left when these have dried up. Owing to the presence of pustules in the mucous membranes, the dejecta and the nasal mucus of a patient may also contain the contagium. But the disease is also contagious in its earlier stages, before the pustules develop, and even, according to a few observations, during the stage of incubation. The variolous poison is certainly very volatile—that is, it is prone to disseminate itself through the air in the neighborhood of the patient in the form of small drops or of fine dry particles of dust. For, in order to catch the disease, it is not necessary to touch the patient, but merely to remain in his vicinity. In many cases we cannot, however, determine with exactness the mode of transmission, since the contagion may either be direct or by means of objects and utensils with which a patient has come in contact—for example, the soiled linen. The dead body also is capable of transmitting the disease. In general, numerous instances point to a considerable "tenacity" in the poison. The precise manner of infection is not yet known. It is most probable that the poison is drawn into the lungs with the inspired air.

It has been demonstrated that the disease can be transmitted to healthy persons by direct inoculation of the contents of the variolous pustules. It is stated that monkeys and other animals may be successfully inoculated in the same way. Whether inoculations with the blood of the sick will reproduce the

disease is not yet settled. The secretions (saliva, sweat, urine, and milk) do not apparently contain the infectious matter.

Course of the Disease. Variola and Varioloid.—The stage of incubation lasts, as a rule, thirteen to fourteen days, sometimes a somewhat shorter time. During this period prodromal symptoms are absent or insignificant.

The disease itself begins suddenly with what are usually very characteristic initial symptoms—rigor, fever, headache, and intense pain in the loins. It is only in comparatively few cases that one or another of these symptoms is slight or wanting. The constitutional symptoms may be very severe—a dry tongue, stupor, wakefulness, delirium. The fever continues intense for some days. The pulse is much quickened. There is almost total anorexia, and often there is vomiting. There is constipation, or, more rarely, diarrhea. Frequently there is a slight sore throat, and sometimes a slight bronchitis. The spleen is enlarged in most of the severe cases, and the urine often has a trace of albumen. In women, menstruation occurs in a remarkably large number of cases. The proper variolous eruption does not at once appear, but from the second day other characteristic efflorescences are not rare. These are termed the initial rash of smallpox. We may find either a diffuse or macular erythema, extending in varying degree over the trunk and extremities (especially on the extensor surface), or a hemorrhagic eruption with small spots, appearing by preference upon the hypogastrium and the inner surface of the thighs (in the so-called femoral triangle of Simon) or on the lateral surface of the trunk and on the upper arm. The erythema soon disappears, but the hemorrhagic spots can be seen longer. Both forms of the initial eruption may be combined.

The initial stage, just pictured, lasts usually three days. Severe symptoms occurring at this time do not exclude the possibility that the further course of the disease may prove favorable, while mild symptoms are of good omen.

At the end of the third or on the fourth day the temperature makes a decided fall, and the regular variolous eruption begins to be developed upon the skin—the *stadium eruptionis*. During this period an evident difference among the separate cases becomes manifest. This distinction cannot indeed be always drawn with a narrow line, but it is noticeable enough to justify the establishment of two types of variolous disease. We refer to the division into a severe form (variola vera), and another, mild form (varioloid). The variola proper has a well-developed eruption with many pustules, and, as a result of this, a second stage of fever (*stadium suppurationis*). Varioloid has a much more scanty eruption, and little or no suppurative fever. We must now discuss these two forms separately.

Variola Vera.—The eruption almost always begins in the face and upon the hairy scalp, appearing somewhat later on the trunk and arms, and last of all upon the legs. It begins in the form of little red dots and spots, which develop in about two days to small papules (*stadium floritionis*). If the hand be passed over thickly set and well-developed papules of variola, a peculiar soft, satin-like feeling is perceived. On the points of these papillæ a little vesicle forms. This keeps growing larger and larger, its contents becoming turbid and purulent, till at last, on the sixth day of the eruption and the ninth of the disease, the development of the genuine pustule of variola is complete (*stadium suppurationis*). The pustule usually presents upon its summit a

little dimple (umbilication), and is surrounded by a red border or "halo." Where the pocks are especially close together, as in the face, the skin between them is diffusely swollen, and the consequent burning and pain are excessive. The countenance becomes much disfigured. Often the eyes cannot be opened because of the œdema. The hands also are apt to be intensely affected, especially the backs of the hands, and also all parts which have previously been injured in any way (pressure or friction of clothing, etc.). On the trunk the pustules are very rarely as close together as on the face and hands. It is worthy of note that often new eruptions of pustules keep appearing for two or three days.

At the same time with the eruption upon the skin, or even somewhat earlier, a perfectly analogous efflorescence develops upon the mucous membranes. The chief places for its appearance are the mouth and throat, the tongue, the soft palate, the nasal cavity, also the larynx, the trachea, and the upper part of the œsophagus. In the vagina and rectum it is rare and scanty. In this mucous efflorescence, however, there are no proper pustules, but small, superficial ulcers. These result from the maceration of the uppermost layers of the mucous membrane. They sometimes become confluent. The annoyance produced by this eruption in the mouth and throat is, of course, very great. The pocks in the larynx manifest themselves by hoarseness, and occasionally by symptoms of stenosis.

As we have said, the beginning of the eruption is the signal for a noticeable fall in the temperature; but in true variola the fall does not reach the normal, or only temporarily. The other symptoms likewise remit, especially the headache and lumbar pain. When, however, the suppuration begins, the fever rises once more, and there are fresh symptoms of constitutional disturbance. This is the time for the dreaded attacks of delirium, during which the patient must be vigilantly watched lest some untoward event happen. Now, too, complications may arise (*vide infra*).

On the twelfth or thirteenth day of the disease the pustules begin to dry up (*stadium exsiccationis*). The purulent contents of the pustules, part of which have burst, form yellow crusts, the swelling of the skin subsides, and, a few days later, the crusts and scabs begin to fall off. With the beginning of desiccation, the fever declines, the local as well as the constitutional symptoms become daily slighter, and convalescence follows. The healing of the pustules is frequently accompanied by an extremely troublesome itching. After the scabs have been cast off, the skin presents pigmented spots, which persist for months. Wherever the cutis vera has itself been destroyed by the suppuration, a scar is inevitable. Thus arise the familiar scars (pitting) of smallpox, which continue visible through life. Very often, after the end of the disease, there is almost complete alopecia. The hair often grows again, but not always.

Varioloid.—The distinction between varioloid and variola vera is not in kind, but in degree. Varioloid is only a milder form of variola. There is, as we have already said, no sharp boundary line between the two. Varioloid is most often observed in those whose susceptibility to the variolous poison has been diminished by vaccination (*vide infra*).

As above mentioned, the behavior of the disease during its initial stage does not permit us to decide positively whether variola or varioloid will be developed. It is true that if the symptoms be especially mild, we may guess that it

will be varioloid; and, likewise, the appearance of the initial erythema already spoken of is regarded as a favorable omen.

Shortly after the pocks begin to appear, the decision can almost always be made with certainty. In varioloid the eruption is rather scanty. It is often irregular, and does not by any means always begin, like that of variola, in the face, but often on the trunk. The individual pocks are in no way different from those of variola; but it often happens that they do not pass through all the regular stages to full suppuration, but undergo resolution before this occurs. Such cases, in which there is nothing beyond papillæ or vesicles, are sometimes spoken of as *variolois verrucosa seu miliaris*. The scantiness of the eruption and the limited amount of suppuration have for their corollary an absence, or at least a very slight development, of the suppurative fever.

When the eruption appears the temperature usually falls by crisis to the normal level and remains there. The desiccation may begin as early as the eighth or tenth day of the disease, so that the whole duration of varioloid is considerably shorter than that of variola. Grave complications are very exceptional. The pocks may develop upon the mucous membranes, but here, too, they are scanty and not very vigorous.

Course of the Fever, Symptoms presented by Separate Organs, and Complications.—1. *Fever* (*vide* Fig. 9).—In the initial stage, as we have said, the temperature rises rapidly, as a rule, with a pronounced rigor; and during the first days it very often reaches 104° to 106° F. (40° to 41° C.). It sinks on

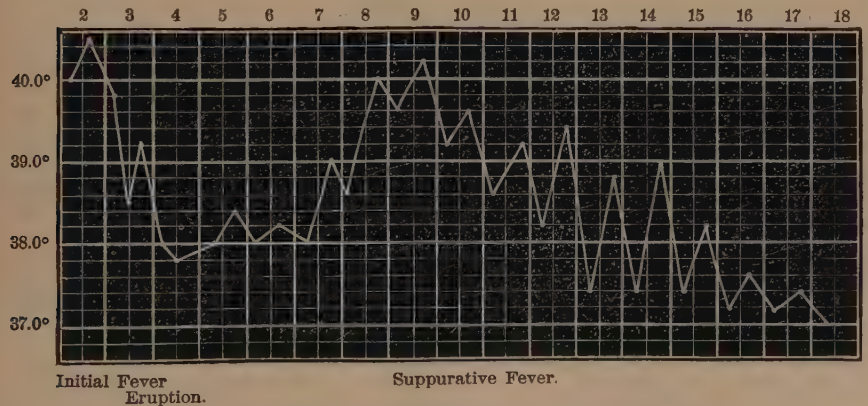


Fig. 9.—Example of the temperature curve in true smallpox.

the third to the sixth day, when the first papillæ develop, and then, in the case of varioloid, falls rapidly to normal, and remains there. In variola the decline is slower and less complete; and with the beginning of suppuration the temperature begins to rise again. The violence of this suppurative fever is usually in direct proportion to the severity of the eruption. It has manifold fluctuations, but seldom lasts, in severe cases, less than a week. Temperatures of 104° F. (40° C.) and higher are common. The fever declines by lysis. In case of approaching death, the temperature may be extremely high, even reaching 108° or 109° F. (42° to 43° C.).

2. *The Skin.*—We have already described the macroscopic appearance of the eruption. It remains to mention briefly the histological phenomena. The

first demonstrable changes are in the cells of the deeper layers of the rete Malpighii. As a result of the variolous infection, the cells perish, are swollen by the lymph which escapes from the papillary blood vessels, and transformed into flaky, homogeneous masses without nuclei ("coagulation necrosis" of Weigert). The lymph becomes more and more abundant, and crowds the cells farther and farther apart. These are thereby finally changed into threads and membranes, forming a distinct network in the vesicle. This explains why, if such a vesicle be pricked, its entire contents are never discharged at once. Great numbers of white corpuscles escape, along with the lymph, from the blood vessels, and finally render the contents of the original vesicle purulent. Proliferative processes occur in the surrounding epithelial cells, which are still intact, and thus the margin of the vesicle becomes elevated, while the dead portion in the center sinks in. Thus the pock becomes umbilicated. If a portion of the papilla itself suppurates, a scar must be left on healing. If the process remains limited to the epithelium, complete regeneration takes place, and the skin reassumes its normal appearance.

Certain secondary complications, which sometimes attack the skin, remain to be mentioned: abscess, phlegmon, erysipelas, gangrene, and bedsores. None of these are due directly to the specific variolous intoxication.

3. *Respiratory Organs.*—The disturbances here are in part symptoms of the specific process of the disease, and in part secondary. The frequent occurrence of secondary symptoms in smallpox is easy to understand (compare the chapter on lobular pneumonia). Of the primary symptoms, we should mention genuine pocks in the larynx, the trachea, and the larger bronchi. As sequels to these, more or less severe secondary disorders are very frequent: laryngeal ulcerations, which may even lead to laryngeal perichondritis and œdema of the glottis; diffuse bronchitis; lobular pneumonia, often of great extent, due to the inhalation of solid matter into the lungs, and frequently accompanied by pleurisy. It should be especially noticed that lobar, croupous pneumonia is not rare. Whether this be likewise secondary, or a direct result of the variolous poison, is not yet known.

4. *Digestive System.*—The genuine pocks often develop, as stated, in the mouth and pharynx, and likewise in the upper part of the œsophagus. They are not observed in the mucous membrane of the stomach or intestines. The active diarrhea sometimes seen depends upon catarrh of the intestine. Dysentery is rare. The eruption in the mouth and throat may result in severe secondary troubles, purulent otitis, parotitis, pharyngeal diphtheria, etc. The spleen is almost always considerably enlarged, and often the liver also, but in a less degree.

5. *Circulatory System.*—Pathological changes in the heart are rare, if we except slight parenchymatous degeneration. Sometimes there is a slight endocarditis (*q. v.*), which is probably secondary. Pericarditis is rather more frequent.

The blood shows a pronounced leucocytosis, mainly affecting the large mononuclear leucocytes, but neutrophilic and eosinophilic myelocytes are also present; the lymphocytes, however, are only slightly increased.

6. *Organs of Special Sense.*—Genuine variolous pustules occur upon the eyelids and the conjunctiva. Later in the disease there may be keratitis, iritis, or choroiditis.

We have already mentioned the relative frequency of aural disturbances, particularly purulent otitis media.

7. *The Joints.*—Articular swelling may appear in the suppurative stage. The shoulders and knees are most apt to be attacked. Periostitis also occurs.

8. *Nervous System.*—We find no pathological changes corresponding to the severe nervous derangements manifested during the disease. After the smallpox is over, spinal diseases sometimes occur, with either paralysis or ataxia. Westphal has shown that they are caused, in some cases, by numerous disseminated centers of inflammation in the spinal cord. Encephalitic processes have also been observed in smallpox, and in some cases neuritic paralysis.

9. *The Kidneys.*—Albuminuria is quite common in severe attacks, but genuine nephritis is a very rare complication.

In pregnant women attacked by smallpox abortion or premature labor is very apt to occur. If a living child be born, it usually dies soon after birth.

Anomalies.—Anomalies in the course of the disease are manifold. We will not speak of the two typical forms already considered. There are abnormally mild cases, with scarcely any initial symptoms, or with an obscure eruption, or with no eruption at all (*febris variolosa sine exanthemate*). In such cases a correct diagnosis is possible only at the time an epidemic prevails, and by the aid of the attendant ætiological circumstances. There are also abortive cases in which the first symptoms are severe, but which recover with remarkable rapidity.

The abnormally severe cases are more important. First, there is the confluent variety. This is merely the typical process in its completest development. The initial symptoms are themselves generally very severe, and are followed, without any considerable remission of the fever, by the eruption of hundreds of pustules. The skin of the face and hands is one continuous area of suppuration. The local discomfort is extreme, as is also the intensity of the fever and of the constitutional symptoms. The nervous system suffers most. There is at the same time an unusually abundant eruption upon the mucous membranes. The occurrence of the above-mentioned complications affecting the various organs of the body is frequent. Death is a common result; or, if recovery takes place, it may be delayed by tedious sequelæ.

Hemorrhagic smallpox is the worst anomalous form. The name is applied to several different varieties. In the first place, any variolous eruption may become more or less hæmorrhagic, and yet the general course of the disease not be essentially altered. Such cases are more common among elderly people, cachectic persons, and drunkards. Secondly, there is a very severe form of smallpox which is generally fatal. The initial stage is marked by the unusual severity of the symptoms. The abundant eruption soon becomes hemorrhagic, and there are also ecchymoses in the mucous membranes and the internal organs. This has been called black smallpox, and by Curschmann *variola hæmorrhagica pustulosa.*

There is another form of hemorrhagic variola, different from these but linked to them by transitional varieties. In it the acute hemorrhagic diathesis develops during the initial stage. Death almost always occurs before the regular variolous eruption. This most frightful form is usually termed *purpura variolosa.* That it is smallpox is proved by its ætiological relations alone. Otherwise it would be impossible to distinguish it from certain other acute

septic disorders. It is prone to attack the youthful and vigorous. Chills, headache, and pain in the loins are the first symptoms, just as in ordinary cases. Cutaneous ecchymoses appear as early as the second or third day. They increase in area so rapidly that one can almost see them grow. They are most extensive in the hypogastric region. There are also ecchymoses in the eyelids, the conjunctiva, the mouth and pharynx, and, as the autopsy discloses, many in the internal viscera. The constitutional symptoms are most severe, and the patient seldom survives the fifth or sixth day of the disease.

Diagnosis.—The certainty with which we can make the diagnosis of smallpox in any well-developed case is equaled by the difficulty of deciding about it during the beginning of the disease, or even during the beginning of the eruption. At this period diagnosis may be impossible. When the variolous eruption is in process of development, it may be confounded with typhus fever, with that form of measles in which the papillæ are prominent, with syphilitic eruptions, and with certain forms of beginning erythema exsudativum. In doubtful cases one must consider not only the cutaneous manifestations but also the other symptoms, for the diagnosis can often only be made with certainty after continued observation. It will require further study to determine the diagnostic value of the inoculation of the rabbit's cornea, and of the demonstration of the Guarnieri bodies (*vide supra*).

Prognosis.—The facts which are of greatest weight in prognosis have already been emphasized. We may repeat that during the initial stage the prognosis of any individual case can seldom be determined. If the first symptoms are mild, or if the initial erythema appears, the case is regarded hopefully. The abundance of the eruption has an influence upon the severity of the disease. Circumstances peculiar to the individual are also important—e. g., age, constitution, or alcoholic habits. We have already called attention to the danger of confluent smallpox, and to the almost absolutely fatal prognosis in the genuine hemorrhagic variety. The mortality varies greatly in different epidemics; on the average it may be taken at about fifteen to thirty per cent. Beyond doubt, the introduction of vaccination has decidedly lessened the fatality of the disease by diminishing the frequency of the severe forms.

Treatment.—1. *Prophylaxis—Vaccination.*—As in all contagious diseases, isolation is of little avail unless complete. This fact has led to the erection in late years of smallpox hospitals. All utensils used by the patient, and his clothing, bedding, and the like, should be most carefully disinfected. The best method is to employ a high degree of heat.

These precautionary measures are employed in many other diseases as well, but for smallpox we are acquainted with a special method of prophylaxis. It is founded upon a fact which is the most remarkable within the domain of the infectious diseases. We refer to prophylactic vaccination. It must long ago have been remarked that a person who has had the disease once, enjoys, to a large degree, immunity from any fresh infection. This suggested the idea of exposing children purposely to contagion, so as to insure them from smallpox for the rest of their lives. The actual inoculation of smallpox is said to have been long practiced in India and China. In the year 1717 it was employed by Lady Mary Wortley Montagu, of England, upon her own son, and with success. Unfortunately, however, the inoculated smallpox proves fatal in many instances; and, being itself contagious, it serves to spread the

disease still further. Then appeared an article written by the English surgeon, Edward Jenner, in 1798. This informed the medical profession of a fact already known to the rural population of his native place, but which Jenner first established scientifically, and recognized in all its importance. There sometimes occurs a disease similar to smallpox upon the teats and udder of the cow, called *variola vaccina*. It is apparently a local trouble, and it can easily be inoculated upon the skin of human beings. Vaccine pustules will be developed upon the spot inoculated. These almost invariably heal without any great constitutional disturbance; but the person vaccinated possesses the same immunity from smallpox as if he had had smallpox itself. This statement of Jenner's was soon confirmed upon every side. The result is the continually spreading custom of prophylactic vaccination.

In some countries, especially in Germany, universal vaccination (compulsory vaccination) is enforced by law; it can be opposed only by ignorance or lamentable prejudice. The discovery of vaccination was the foundation for all the subsequent remarkable discoveries in immunity and in serum investigation. It has been definitely proved by Fischer and others that vaccinia is, in fact, only a form of smallpox rendered milder and modified by transmission to animals. If we inoculate a calf with the contents of a fresh smallpox vesicle from man, typical vaccinia is produced. With the contents of this vaccine vesicle children can be inoculated without any danger, and they are no longer susceptible either to vaccinia or smallpox.

We can mention only the most important of the details relating to vaccination and the method of its performance. Only exceptionally do we vaccinate with the contents of a human vaccine vesicle ("humanized lymph"), but we almost invariably use animal lymph from vaccine vesicles of calves. This is now obtained in large quantities and put on the market. The process of vaccination is to make three shallow incisions, 3 or 4 cm. apart, in the skin of the upper arm, and to introduce the vaccine lymph into them. The surrounding tissue becomes swollen in three or four days. In seven or eight days the vaccine vesicles are well developed, if the disease takes its normal course. Next they become purulent, and then dry up, and finally, on healing, leave the familiar scar behind. The whole process occupies about three weeks. If the vaccination fails, or is but partially successful, it must be repeated after a few months. The protective power of vaccination does not last indefinitely, and therefore revaccination is necessary every five or six years. The first vaccination of children usually takes place when they are three or four months old. If they are feeble we wait longer, unless smallpox is prevalent.

According to the vaccination laws of Germany every child must be vaccinated before the end of his first year of life, and all school children must be revaccinated during their twelfth year.

It must be confessed that vaccination is not without its dangers. The little cutaneous wound made by it may lead, like any other, to sepsis, or to erysipelas. The latter has been called vaccination-erysipelas. But such misfortunes are extremely rare. The not uncommon "vaccine roseola" deserves especial mention. It appears first upon the arm vaccinated, and spreads over the rest of the body; but it is not a serious matter. It is, of course, possible, by especially bad luck in the use of human lymph, that other diseases, among which syphilis is of chief importance, may be inoculated along with vaccinia;

but this is a very rare occurrence—much more so than the enemies of vaccination pretend. If the physician exercise proper care in the selection of the person from whom to take humanized virus, it can be entirely avoided.

Impetigo contagiosa and favus have been observed as the result of transmission by vaccination, but they have no serious significance.

[The incubation stage of vaccinia being shorter than that of smallpox, the prompt vaccination of an unprotected individual who has been exposed to infection should always be practiced, if possible; oftentimes the severe disease may thus be prevented.]

2. *Symptomatic Treatment.*—The treatment of smallpox is purely symptomatic. When the disease has once begun it is too late for vaccination to have any influence upon its further course. During the initial period we may advantageously employ cool baths to diminish the fever and alleviate the constitutional symptoms. An ice bag will relieve the headache. We must not let the lumbar pains lead us to any but a cautious use of local irritants, for the pocks come out in greater abundance upon such portions of the skin as have been in any way irritated. If the disease proves, during the stage of eruption, to be varioloid, there will be no further need of special treatment. Good nursing and proper food will suffice.

The true smallpox, on the other hand, demands the interposition of the physician. He must strive to guard the regular course of the disease in the skin and in those portions of the mucous membrane which are accessible from being disturbed by secondary inflammations; for we have no doubt that the ruptured pustules furnish a most easy ingress to septic impurities from the surrounding atmosphere, so that later, when there is extensive suppuration of the skin, or analogous and severe disturbance in the mucous membrane, it is impossible to discriminate between the effects of the smallpox itself and those due to the secondary suppuration. If we were able to have the whole process go on "aseptically" we should certainly have made an important advance in therapeutics. It is very doubtful if this is to be attained by means of externally applied disinfectants, such as the paste recommended by Schwimmer (acidi carbol., 4 to 10 parts; olei olivarum, 40 parts; cretæ opt. trit., 60 parts; to be spread on linen and applied to the affected surfaces). Many physicians, therefore, confine themselves to the use of cold applications, Priessnitz packs, simple salves and oils. Under Hebra, in Vienna, continuous warm baths were employed in severe cases with great success. The recent claims of various observers that the red-light treatment (i e., the sick-room so arranged that only red light can enter) has a decidedly limiting effect upon the suppuration requires further substantiation.

The treatment of the affected mucous membrane in smallpox must also meet the indication above mentioned. The most thorough disinfection of the mouth and pharynx must be aimed at. The means to be used are careful washing and gargling with solutions of chlorate of potash (1 to 30), borax, permanganate of potash, hydrogen peroxid, etc. The eyes, if they need it, must also be appropriately treated. As to all other complications, cool baths are relatively the most useful remedy. They can be given without difficulty. The chief indications for them are severe pulmonary or nervous symptoms, or continuous high fever. Internal antipyretics, such as quinin or antipyrin, are also employed. Violent nervous disturbances, such as delirium, sometimes

require the cautious use of narcotics. There is nothing to add as to the treatment of malignant hemorrhagic smallpox, for, as we have said, such cases are unfortunately almost hopeless.

CHAPTER VIII

VARICELLA

(Chicken Pox)

Varicella is truly one of the diseases of childhood. Adults very rarely have it. It is contagious, and often comes in epidemics.

The stage of incubation does not last over thirteen to seventeen days. The disease begins with the appearance of vesicles, the size of a pea or a little larger, usually having a small red areola, and varying from ten to one hundred or more. The trunk usually bears the greater part of the vesicles, while the extremities have few. The face is frequently the seat of a considerable number, and there are often a few upon the hairy scalp. There may be a vesicle here and there upon the mucous membrane of the mouth or palate. There are seldom any prodromata. Slight symptoms of fever may attend the eruption itself. The full eruption does not appear at one time. Successive crops of vesicles develop over a period of several days or of even almost one week. For this reason one sees, at one and the same time, early, fully developed, and healing vesicles. The contents of the vesicles always become slightly cloudy (purulent), but the cutaneous tissue is almost never deeply involved as in smallpox. The course of the disease is generally completed in from one to two weeks. Most children feel perfectly well the whole time, although in rare cases there may be pain in the limbs, anorexia, and a slight coryza. Severe complications are seen very exceptionally, the most frequent one being a mild nephritis.

Exceptionally, the disease may be rudimentary, with a varicelloid roseola and no formation of vesicles. On the other hand, some cases present quite severe constitutional symptoms and a high fever, even reaching 105° F. (41° C.) temporarily. In most cases, however, as we have said, the child is so slightly disturbed that a physician is hardly thought necessary.

Diagnosis.—The diagnosis is almost always easy. Formerly varicella was often confounded with smallpox, and for a long time the followers of Hebra, in Vienna, for some inconceivable reason, maintained the identity of the two. That they are essentially distinct is shown (1) by the epidemics of the two appearing entirely separate from each other, (2) by the fact that having one does not give immunity from the other, and (3) by the uniform failure of attempts to produce variola by inoculating varicella, or *vice versa*.

Prognosis.—The prognosis is perfectly good. There is usually no special treatment necessary, but young children and those having fever should be kept in bed till the eruption has dried up.

CHAPTER IX

ERYSIPELAS

(*St. Anthony's Fire*)

Ætiology.—Erysipelas is a peculiar inflammation of the skin, recognized by redness, swelling, and pain. It has the peculiarity of spreading gradually, by direct extension, from its point of origin over a larger or smaller portion of the body. The cause of this inflammation, as was first shown by Fehleisen, is a local infection by the streptococcus pyogenes and its further extension in the skin. The exciting agent of erysipelas is then bacteriologically identical with the streptococcus which is the cause of phlegmonous suppuration, severe septic infection, etc. The reason why in the individual case erysipelas develops, and not some other streptococcus disease, is the special variety of infection and the further spread of the streptococci in the deeper lymphatics of the skin, and in part perhaps the special "virulence" of the infecting germs.

The former distinction between "traumatic" and "idiopathic" or "exanthematic" erysipelas can no longer be rigidly maintained. Every erysipelas is in a certain sense traumatic, since infection with streptococci cannot occur through the unbroken skin. The so-called "idiopathic" erysipelas is distinguished from "wound erysipelas" (in which, of course, we include puerperal erysipelas, erysipelas of the newborn starting from the umbilical wound, etc.) only by the fact that in the former the infection takes place in small, insignificant wounds of the skin which are easily overlooked. Such an erysipelas occurs chiefly in the face and head, and the following description of the symptoms refers chiefly to this facial erysipelas. Most of these cases of erysipelas arise from little excoriations, cracks, and scratches about the nose or ears, or more rarely about other parts of the face or scalp. A previous coryza, by the accompanying slight erosions about the nose, sometimes gives the opportunity for an attack of erysipelas, but sometimes the infection occurs in the mucous membrane of the nose or pharynx; then we have a primary erysipelas of the mucous membrane, which extends through the nasal passages and finally appears on the external surface of the nose.

FIG. 10.—The cocci of erysipelas. ×700. Section through a lymph-vessel in the skin.

Facial erysipelas is most apt to attack the young, and seems to be somewhat more frequent in women than in men. The laity erroneously regard catching cold and getting frightened as frequent causes of the disease. If we except the predisposing causes above mentioned—viz., coryza, slight scratches, cuts, etc.—we usually find no cause of which we can feel certain. Often

endemic influences are important. It has been long known that traumatic erysipelas can get so secure a footing in particular hospitals or wards that every wounded person treated in them is in danger of this disease; but the apparently idiopathic variety is sometimes remarkably frequent in particular places (barracks, etc.). Several members of one family may likewise have facial erysipelas simultaneously. In nearly all such cases the sufferers are infected from some common source, for direct contagion is certainly exceptional; but patients with open wounds should never be put near erysipelas cases, because here the risk of infection cannot be wholly excluded. Direct inoculation can, however, as has been proved, convey the disease from a patient to other persons or to animals.

In contrast with the behavior of many other infectious diseases (typhoid fever, measles, scarlet fever, smallpox, etc.), erysipelas is peculiarly apt to attack the same individual over and over again. There are persons who have facial erysipelas about every one or two years, and ten to fifteen times. Often the explanation of this apparently lies in some chronic disease—e. g., chronic ozæna, chronic eczema of the nostrils or ears—which makes infection easy, but in other cases no cause can be discovered. Of course when erysipelas afflicts the same person a number of times the individual attacks gradually become milder. Marasmus seems to predispose to erysipelas. At least we have observed that erysipelas occurred with relative frequency, in the Leipsic hospital, in patients suffering from the last stages of phthisis or cancer, or similar diseases.

Clinical History.—The incubation period of erysipelas is short, generally lasting but from one to three days. In many cases the first subjective symptoms are simultaneous with the cutaneous swelling, and these are chiefly local. There is pain and a sense of tension in the skin. Soon subjective symptoms of fever also appear, such as general malaise, anorexia, headache, and sometimes vomiting. In other cases the disease starts with more violent constitutional symptoms: there is an initial rigor, with violent headache and great languor. The local erysipelatous swelling appears almost at the same time with these general symptoms, or sometimes a few days later, either because the local inflammation is at first slight or because it is not noticed, as in the scalp when the hair is very thick. In rare instances the disease begins with sore throat. We saw three almost simultaneous cases of facial erysipelas in one family, in which a severe sore throat lasted for four or five days preceding the appearance of the cutaneous disorder. We have seen a number of similar cases since, and believe that they are due to a true erysipelatous sore throat—that is, to an erysipelas of the soft palate which extends through the nostrils to the external skin. In cases of erysipelas which follow a coryza we may assume, as stated above, that there sometimes is an erysipelas of the nasal mucous membrane preceding the erysipelas of the skin.

The erysipelatous process in the skin is almost always circumscribed at first. It usually starts on the nose, less often upon the cheek, the ears, or the hairy scalp. The skin becomes considerably swollen, grows red, smooth, and shiny, and feels hot. The redness and swelling keep spreading. There is usually a sharp, elevated ridge, perceptible to sight and touch, separating the diseased from the still healthy portion of the skin. As long as the erysipelas is spreading, we see stretching out from its border, or somewhat removed from

it, small red streaks and spots which gradually increase in area and intensity, and finally coalesce. Any decided fold in the skin may hinder for a time the extension of the disease. The nasolabial folds are particularly apt to limit it. The border of the hairy scalp frequently forms a terminal line; but the whole scalp may be attacked, the inflammation stopping only when it reaches the nape of the neck. The boundary of the erysipelas in the hairy scalp can usually be quite accurately determined by palpation (swelling and local tenderness).

It is only in a relatively small number of cases that it spreads farther yet, attacking the back, the arms, and the anterior surface of the trunk, or even extending to the feet. This is known as *erysipelas migrans*. The facial erysipelas may be healed long before the disease ceases to extend over the other parts of the body. When the spreading process is about to cease, the inflammation usually becomes decidedly milder, appears only in isolated spots, and finally stops completely. In most cases, only the face, the ears, and a part of the scalp are attacked.

It is not a rare thing for vesicles or bullæ to form in the portions of skin attacked. Such cases are called *erysipelas vesiculosum* or *erysipelas bullosum*. The serum may change to pus in these blisters, and then we have *erysipelas pustulosum*. Exceptionally the infiltration of the skin becomes so intense as to result in a localized necrosis or gangrene—*erysipelas gangrænosum*. The parts most apt to be attacked by this are the eyelids, where abscesses sometimes form.

Microscopic examination of the skin shows a marked hyperæmia of all the blood vessels and a very considerable infiltration of both the skin and the subcutaneous connective tissue with serum and cells. In those parts where vesicles are formed there are many dead and disintegrated epithelial cells in the rete Malpighii. In the parts where most streptococci develop there may also be necrosis of the deeper layers of the skin. We have already said that there may be many streptococci in the lymphatics of the skin, but they are found only in the first fresh stages of the inflammation. When the inflammatory exudation has reached its full height, the streptococci have usually wholly disappeared. As a rule, we find only a few or no streptococci in the contents of erysipelas vesicles.

The inflammation in any one spot usually ends four or five days after it has made its appearance there. There is apt to be much attendant desquamation. The face is often left with a finer complexion than it had before.

The other symptoms, of which the constitutional disturbance and the fever are chief, correspond pretty closely to the severity and extent of the cutaneous lesion. It is comparatively seldom that this correspondence does not exist.

The fever in facial erysipelas usually rises rapidly at first, and to a considerable height. We have seen but few cases where the high fever was delayed till a day or two after the skin was attacked. The temperatures observed in erysipelas are often extreme: 106° F. (61° C.) is not at all rare. The highest we ever saw was 107.2° F. (41.8° C.). While the erysipelas continues or is spreading, the fever is seldom continuous, nor are the remissions insignificant. Pronounced intermissions, even down to normal, are very frequent, but are followed again by a rapid and great rise of temperature. The fever may terminate with a genuine crisis (see Fig. 11). In intense cases of con-

siderable extent, or in erysipelas migrans, the termination is more apt to be by a more or less gradual lysis. We have seen the cutaneous inflammation in erysipelas migrans still extend itself a little, in a rudimentary form, after the fever had completely ceased.

The headache is often intense, and seems to result not merely from the inflammation of the scalp but from disturbances of the circulation in the underlying parts, or more probably from the action of a toxin. Other severe cerebral symptoms are also relatively frequent. The patient may be very restless, excited, and wakeful. At night there may be mild or even violent delirium; or there may be decided stupor. All these symptoms are in chief part due to the general intoxication caused by the infection; but they also justify a surmise, as we have said, that there is a circulatory derangement in the meninges and the brain itself, resulting from the inflammation of the scalp. In drunkards, delirium tremens is not infrequent.

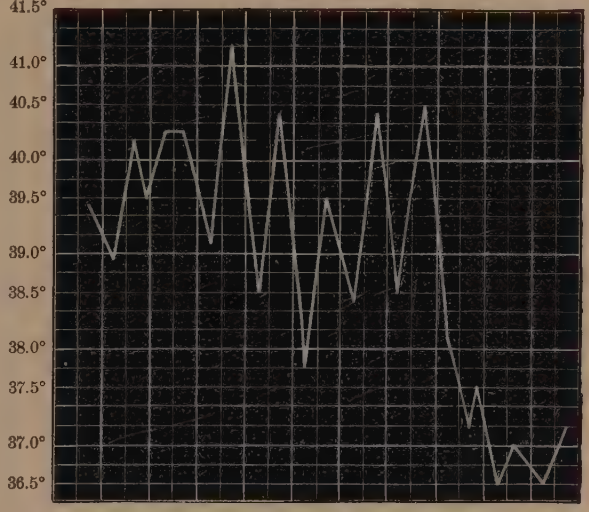


FIG. 11. — Example of the fever curve of a case of severe facial erysipelas (Erlangen Medical Clinic).

One of the most constant symptoms in facial erysipelas is gastric and intestinal disturbance. There is usually complete anorexia. The tongue is thickly coated. Vomiting is frequent, not only at the beginning but during the course of the disase. There is constipation, or there may be quite severe diarrhea. The spleen is usually moderately swollen.

In almost all severe cases the urine contains albumen, some casts and white blood cells. In most cases we find in the blood a pronounced leucocytosis (16,000 to 18,000 leucocytes in a cubic millimeter). Streptococci are not usually found in the blood.

The entire duration of the disease varies greatly in different cases. A very light case may get well in a few days. Most cases of average severity last a week or ten days. Erysipelas migrans may continue for many weeks. We have several times seen a relapse come on after a number of days of complete apyrexia. Either the face would be once more attacked, or some portion of the skin which had previously escaped.

Complications.—Local complications are comparatively rare and insignificant in erysipelas. The lymphatic glands of the throat and back of the neck are very frequently somewhat swollen, but seldom attain great size. Bronchitis and lobular pneumonia may develop in severe cases, but are not at all characteristic. Some observers call attention to the occurrence of pleurisy, endocarditis, and pericarditis; but these complications also are very rare.

Sometimes there is an icteroid hue. As stated above, the urine frequently contains a small amount of albumen, and in severe cases of erysipelas acute hemorrhagic nephritis is not so very rare, but it almost always disappears entirely. The albuminuria usually disappears a few days after the fever. Swelling of the joints has been repeatedly observed. It is more frequent in the severe surgical forms of erysipelas, which are combined with universal septic and pyæmic conditions of the system. Purulent meningitis may complicate an erysipelas located in the head, but it is very rare. We should be exceedingly cautious about asserting its existence even when the cerebral symptoms are very pronounced.

Cutaneous complications are relatively frequent. We have seen herpes labialis quite often, and a number of cases of urticaria. Of much greater importance are the cutaneous abscesses which occur in severe cases. These are due to a phlegmonous or even gangrenous inflammation of the connective tissue. Their most frequent seat in the face is the eyelids, as already stated; and in that case the eye may itself be endangered. At the close of severe cases of erysipelas migrans, numerous abscesses may develop in the skin of the trunk and extremities, delaying convalescence. Since, as we have said, the streptococcus of erysipelas is absolutely identical with the streptococcus pyogenes, all purulent inflammatory complications of erysipelas are to be referred to the local or metastatic action of the original infectious germs, but erysipelatous nephritis is probably of purely toxic origin.

Diagnosis.—The diagnosis of erysipelas is almost always easy when once the cutaneous lesion has developed. Phlegmonous inflammation of the skin and lymphangitis are to be eliminated; but this is always possible, with proper care. After a single examination, we may confound it with acute facial eczema of great severity, or even with a marked urticaria. Chief attention should be paid to the characteristic border of erysipelas and to its manner of extension. In erysipelas beginning in the hairy scalp the local inflammation may easily be overlooked, being completely masked by the severe general symptoms.

Prognosis.—The prognosis of facial erysipelas, when it attacks a healthy person, is very favorable. In drunkards a severe case may be complicated by delirium tremens, and the issue be unfavorable. We saw one case end fatally because of gangrene of the eyelids, followed by purulent inflammation of the orbital connective tissue. Erysipelas migrans may so exhaust the powers of feeble patients as to become dangerous. The prognosis of surgical erysipelas is relatively more unfavorable, but it cannot be considered here.

Treatment.—The treatment of ordinary erysipelas of the head and face, which is the main question here, is purely symptomatic. The large number of remedies recommended (tincture of iodin, nitrate of silver, iodoform, ichthyol, carbolic acid, collodion, etc.) shows that they cannot any of them materially influence the morbid process. It is easy to be deceived about their efficacy, as most cases are relatively benign. We therefore usually content ourselves with powdering the affected skin or covering it with oil or vaselin to relieve the tension, and also with putting on an ice bag if the patient likes it. We can expect no specific action from internal remedies (Pirogoff's camphor cure, liquor ferri sesquichlorati, etc.).

Apart from alleviating the local discomfort, some other symptoms occasionally demand special consideration. Severe headache and other severe nervous

symptoms may be relieved by the local use of cold or by antipyrin and similar remedies. With high temperature cool baths or antipyretics internally may be used with benefit, but in general the fever does not often demand energetic treatment, since in erysipelas, as we have said, considerable spontaneous remissions of temperature often occur. When there are marked gastro-intestinal symptoms we may prescribe hydrochloric acid, opium, etc.

Only when there is a true migrating erysipelas and the disease spreads over the body should we try more energetic methods. Strips of adhesive plaster, firmly applied at the border line of the lesion, may be tried; thereby the lymph vessels may be compressed, and the spread of the inflammation thus interfered with. Hueter recommended subcutaneous injections of a two-per-cent solution of carbolic acid a little way from the edge of the erysipelas. We have seldom seen great benefit from any of these methods. Better results are promised from the plan recently advanced to scarify the erysipelatous area, and then apply a sublimate lotion. Many attempts have been made to produce an effective antistreptococcic serum, and sanguine therapeutists believe they have obtained curative results from the injection of some such sera. I do not wish to advise against such attempts being made, but proof of the effectiveness of the serum treatment of this disease is entirely wanting. [Johnson, of Toronto, reports favorable results from vaccination with devitalized cultures of streptococcus erysipelatis.] In the severe cases the main point, after all, is to maintain the patient's strength by nursing and food. If cutaneous abscesses form, they should be opened promptly, when they usually soon heal.

CHAPTER X

DIPHTHERIA

(Diphtheritis. Croup. Cynanche contagiosa)

Ætiology and General Pathology.—Clinically, "diphtheria" means a certain well-characterized, specific, acute, infectious disease, the chief visible lesion of which is a croupous-diphtheritic inflammation of the pharynx and upper air-passages. In a purely pathological sense, however, the terms "croupous" and "diphtheritic" have a broader meaning. They denote a certain form of inflammation which may occur in the mucous membrane of almost any part of the body. It is frequent in the intestine and bladder. There is great diversity in the causes which may produce it.

The pathological characteristic of every croupous-diphtheritic inflammation consists in the formation of a fibrinous exudation. This may either be a croupous membrane, which is grayish white, rather firm and elastic, and which can be lifted off with comparative ease from the mucous membrane upon which it rests, or it may be a diphtheritic infiltration with necrosis of the tissues. Here the exudation is more or less deeply imbedded within the proper structure of the mucous membrane itself. There is no essential difference between croup and diphtheria; diphtheritic inflammation is the severer form of the disease, croupous inflammation the milder. In diphtheria the fibrinous exu-

dation is preceded by a necrosis of the epithelium and of the underlying tissues of the mucous membrane as well, while in the case of croupous exudation the necrosis is limited to the epithelium. The croupous membrane never rests upon an intact mucous surface, but replaces the epithelium, which has already been totally or in very large part destroyed. Flaky remnants of the epithelium, no longer nucleated, are sometimes found in the meshes of the fibrin. The preceding destruction of the epithelium is essential to the occurrence of fibrinous, croupous inflammation. The fibrinous exudation can be formed in those places only where the cause which excites the inflammation kills the epithelium at the same time. It is still undecided whether or in what degree the epithelium takes part in the formation of the croupous membrane. Most pathologists hold that the material for the fibrin comes from the fibrinogen of the inflammatory matter which transudes through the walls of the vessels, and also from the disintegrated migratory white blood globules. These last are abundant throughout the deposit itself, and still more numerous in the entire tissue of the mucous membrane beneath the croupous or diphtheritic exudation. If recovery takes place in croup, all that is needed after the exudation has been cast off is the renewal of the epithelium, which can be accomplished through the exclusive agency of the remnants of epithelium left along the borders of the diseased spot. In diphtheria, however, the entire necrotic portion of mucous membrane must slough off, a line of demarcation being formed, and cicatricial tissue replaces the necrosed portion.

The above is a bare outline of the present views about croupous and diphtheritic inflammations. They have been reached gradually through the labors of E. Wagner, Weigert, and others. We have not yet touched upon the ætiological factors; but what precedes renders it evident how manifold they may be, for many causes which destroy the epithelial layer of the mucous membrane, and at the same time promote inflammation, may excite croup. We have mechanical causes, such as impacted feces, gallstones, renal calculi; chemical irritants, caustics, like ammonia and the acids; and, finally, a number of specific, infectious, disease-producing poisons. Among these is the specific poison of diphtheria proper.

There has been no doubt for a considerable period that true pharyngeal diphtheria could be produced only by a specific organized cause. To demonstrate this, however, was extremely difficult, for in the diseased parts there are a great number of diverse microörganisms, originating in the mouth and throat, and really secondary to the diphtheritic process, that were very hard to distinguish from the diphtheria bacilli. It was only in 1883 that Löffler was able to demonstrate that the specific diphtheria bacillus is a form of bacillus characterized by a definite and particular granular formation, a club-like swelling at the ends, and special methods of growth on blood serum. (*Vide* under "Diagnosis.") Löffler's observations were substantiated on all sides.

The diphtheria bacilli are almost always found only on the diseased portion of the mucous membrane or the point of inoculation, and never in the blood or the internal organs. This fact is of the greatest pathological significance, and it has been established not only for diphtheria in man, but also for experimental diphtheria in animals. In pharyngeal diphtheria they are found chiefly on the under surface of fresh false membrane. The severe general symptoms of diphtheria, however, except when due to secondary infection

(*vide infra*), depend upon the poisonous chemical action of certain substances produced by processes of metabolism in the bacilli.

We may accept it as a fact that the diphtheria bacilli almost always reach the pharyngeal or nasal cavity directly through the inspired air or in some other way, and there, in case they attach themselves to the mucous membrane and increase, produce diphtheria. In a few rare cases they seem to be inhaled into the larynx at once, and to excite there a "primary laryngeal croup" (*vide infra*). The source of the infection is always to be referred ultimately to another case of diphtheria, but the way in which the disease spreads cannot always be traced in detail. In many cases, of course, a direct ("contagious") transmission of the poison is evident, as from coughing, or the many cases of the disease in physicians and nurses from sucking the croupous membrane out of the tracheotomy tube in children, etc. If several children in one house fall sick, as often happens, we may here suspect a direct transmission of the disease, although it is also possible that in such an event several cases may arise from the same source of infection. It seems certain that the infection can be carried from one person to another by some intermediary, by clothing, linen, toys, and other objects to which diphtheria poison clings. The diphtheria bacilli show considerable resistance to external influences (temperature, drying, and the like) when they have dried in rather thick masses and are shielded from light. For this reason, diphtheria bacilli on the walls or floors of damp and unwholesome dwellings may retain their virulence for a long period. After apparently complete recovery from diphtheria, virulent diphtheria bacilli may be found in the oral cavity for a considerable time.

FIG. 12.—Diphtheria bacilli (Löffler's methylene blue).

FIG. 13.—Diphtheria bacilli (Neisser's polar body stain).

This is particularly to be noted on account of its practical importance. Indeed, diphtheria bacilli have been found in healthy individuals who have been in contact with diphtheria cases ("bacillus carriers"). We must mention, in conclusion, that attention has lately been called to the possibility of the transmission of diphtheria from sick animals (hens, doves, calves, [cats]) to man, since diseases resembling diphtheria undoubtedly occur in the domestic animals mentioned. It is, however, very unlikely that the disease can be thus transmitted, for, as Löffler has shown, the bacilli in hens and calves are entirely different from the human variety.

Diphtheria, as is well known, is a disease chiefly of childhood, but some cases, and even very severe ones, may occur in adults. In advanced life the disease is very rarely seen. In large cities sporadic cases occur from time to time, but the disease often takes on an epidemic character.

[While it seems in the highest degree probable that the poison is usually purely local at the start, cases occur which suggest that constitutional infection through the pulmonary blood vessels may precede the local manifestations. Infection through the alimentary canal is not probable, though its occurrence cannot be positively denied.

There are still points in the ætiology and pathology of this affection which are involved in obscurity. Much has been said and written in this country and in England about the relations of filth and diphtheria. That filthy surroundings contribute a soil favorable to the development of the poison, and at the same time diminish the resisting power of the human organism, cannot be doubted; but, as long as the parasitic theory of infectious diseases prevails, sewer gas and the like must be classed among the predisposing or accessory causes.

Some of the frightfully virulent epidemics of diphtheria in sparsely settled country districts and on the Western plains are difficult to explain under the theory that each case is mediately or immediately the result of a previous case; these difficulties will, however, doubtless be cleared away in time.]

Clinical History.—The incubation is rather brief, seldom exceeding two to five days. The disease itself almost always begins with general malaise, headache, fever, and pain on swallowing. Little children, however, often do not complain of this last symptom, and in older children the sore throat may not be very troublesome at first. It is therefore a very important rule for the physician to examine the throat carefully in every child who presents ill-defined general symptoms. If diphtheria is beginning, we find redness of the soft palate, and more or less swelling of the tonsils. Upon the inner surface of the latter, and perhaps upon the arch of the palate and the uvula also, are spots covered with a grayish-white coating, which is quite firmly adherent to the mucous membrane. They are less frequent at first upon the posterior wall of the pharynx and the hard palate. Their extent varies greatly in different cases. In the mildest they are chiefly confined to the tonsils, attacking the soft palate or the tonsillar surface of the uvula but little if at all. In severer attacks the spread of the false membrane during the first days of illness is rapid. Almost invariably there is a very early and considerable swelling of the lymph-glands at the angle of the jaw. The constitutional symptoms persist. The children are restless. There is complete anorexia, and frequently vomiting. The temperature curve is not typical. It is irregular, but is often rather elevated. In true diphtheria such high temperature, 104° to 105° F. (40° to 41° C.), as is common in follicular tonsillitis, is certainly exceptional. On the other hand, fever may be slight or almost absent, even in the worst cases. The pulse is very rapid. The urine may have a trace of albumen and sometimes more. Herpes labialis is occasionally present.

In mild cases the local and constitutional symptoms remain moderate; and at the end of a week or ten days there is decided improvement, with rapid convalescence. In severe cases, however, dangerous symptoms appear, perhaps early; the croupous inflammation involves neighboring organs, or a severe constitutional condition is developed.

The diphtheria very frequently extends into the nose. This, though not in itself dangerous, is usually a sign that the case is a severe one. The inflammation of the nasal mucous membrane may be simply mucopurulent, but it

may also be croupous. It is betrayed by the abundant purulent discharge. Excoriations and superficial ulcers are usually soon produced at the edge of the nostrils. There may be nosebleed.

A much more dangerous complication is the extension of the croupous inflammation into the larynx. This creates a mechanical hindrance to respiration, which proves fatal in a great many cases, as the child's larynx is so small. Formerly "croup"—i. e., croupous inflammation of the larynx—was regarded as a different disease from diphtheria. Many specialists in children's diseases still maintain this view; but it is in direct opposition to all anatomical, clinical, and ætiological facts. We grant that there are cases where the pharynx is slightly affected, while the croupous inflammation of the larynx is extreme; and once in a great while the diphtheritic infection results in croupous laryngitis and tracheitis alone, the pharynx escaping disease. Still, the proposition that there are two distinct diseases, "croup" and "diphtheria," is absolutely untenable. In the overwhelming majority of cases the throat is first affected and then the larynx. We should also consider how easily slight lesions in the pharynx may be overlooked, especially if located upon the posterior surface of the soft palate or upon the epiglottis. Cases of what is called "ascending croup," in which the laryngeal affection precedes the appearance of the disease in the pharynx, are, to say the least, very exceptional.

Hoarseness is usually the first indication that the diphtheria has attacked the larynx. Then follows the peculiar, harsh, ringing, "croupy cough," so dreaded by the parents, and, finally, there are signs of beginning laryngeal stenosis. Respiration is not much accelerated, but is labored, and the accessory muscles of respiration are called more and more into action. The child becomes more restless and anxious. Its face grows pale and livid. Even in children who can make no definite statement as to their subjective sensations these symptoms which we have mentioned are very noticeable. The chief cause of the dyspnœa is undoubtedly the mechanical stenosis due to the croupous deposit. Paralysis of the laryngeal muscles may perhaps be a factor. If portions of the false membrane become partially detached, they may act like valves, being sucked in at each inspiration, and pushed aside by the current of expired air. If greater stenosis occurs, respiration becomes noisy, resembling snoring. Inspiration, particularly, is prolonged and "sawing," and is attended by marked depression of the larynx toward the sternum. An important diagnostic point is the drawing in during inspiration of the suprasternal region, the epigastrium, and the lower part of the sides of the thorax. This is the direct result of the obstructed flow of air into the lungs. As the lungs do not expand enough to correspond to the inspiratory dilatation of the thorax, the parts mentioned are forced in by atmospheric pressure. The degree of dyspnœa may vary at different times. The false membrane may be loosened and coughed up, rendering respiration easier for a time, till fresh exudations or displacements of membrane cause renewed distress. Spontaneous recovery is still possible. The membrane is expectorated and no more formed. In most cases the symptoms of suffocation increase more and more, respiration grows quicker and more superficial, and the child becomes more and more stupefied by the excess of carbonic dioxid in the blood. The pulse gets very small, rapid, and irregular. There are mild convulsions and then death. The

autopsy in these cases usually discloses that the croupous inflammation has extended into the larger bronchi or even into the smaller. The lumen of the bronchioles may be almost completely occluded by false membrane. This croupous bronchitis gives rise to no especial auscultatory phenomena during life, so that often we suspect it rather than diagnosticate it. Its clinical significance, however, is very great, since, even in adults, it may cause death by suffocation without any especial stenosis of the larynx. If it is present, respiration may not be materially relieved in spite of tracheotomy.

Besides the dangers attendant upon the local extension of the diphtheritic inflammation, the general intoxication of the body must be regarded as the second most important circumstance in our general estimate of the disease. As mentioned above, the diphtheria bacilli produce an extremely poisonous toxin, which enters the circulation and which may give rise to the severest symptoms. Indeed, special attention must be called to the fact that the general symptoms are not infrequently very slight, in spite of extensive local croupous inflammation. On the other hand, where the local disease is relatively slight, severe general symptoms are sometimes to be observed which may justly be referred to diphtheritic intoxication. Among these severe general symptoms are somnolence, which may increase to complete sopor, an increase in the pulse to 120 or 140, progressive cardiac weakness, and collapse. The conditions are different in those cases which are termed septic or malignant diphtheria. Here the local disease in the pharynx usually has a special appearance: instead of the croupous deposits we see a deeper-seated necrotic destruction of mucous membrane covered with a foul-smelling, greasy, purulent deposit ("gangrenous diphtheria"). In such cases the lymph-glands in the neck are almost always much swollen. The tongue is dry and fissured, and a stinking secretion runs from the nose. The temperature is usually not very high, but the pulse is very rapid and small. In some of these cases there is certainly a secondary septic infection, probably in most cases from streptococci, as in scarlatinal diphtheria (*vide supra*).

It is to be noted, however, that sometimes diphtheria bacilli with their toxins are alone capable of producing such septic clinical pictures. In fact, Heubner and others deny that mixed infections play any rôle whatsoever in cases of septic or malignant diphtheria. They believe that the diphtheria bacilli are alone responsible for every symptom present. In my opinion, this viewpoint is somewhat defective.

Regarding the manifestations in other organs in diphtheria, we have already said that the nose, larynx, trachea, and bronchi are often involved, but the dense squamous epithelium of the œsophagus and the mucous membrane of the stomach, which is protected by its secretion, are very rarely affected. I have, however, seen two cases of cicatricial œsophageal stenosis following diphtheria. Diphtheria not infrequently extends to the Eustachian tube and middle ear; more rarely to the oral cavity, the lachrymal canal, and the conjunctiva. The latter may also be affected by the direct conveyance of the infectious matter by the finger, etc. In a similar way diphtheria may sometimes involve the external genitals (in girls) or any accidental wounds or injuries of the skin, such as blistered surfaces, etc.

Of the internal organs, the lungs, heart, and kidneys demand chief attention. In the lungs, besides croupous bronchitis, pneumonia often develops in

severe cases. This is usually a lobular catarrhal pneumonia, more rarely lobar croupous pneumonia. Although we cannot absolutely deny that the pneumonia may sometimes be of a true diphtheritic nature, the majority of cases of pneumonia developing in diphtheria must certainly be classed as secondary affections, due to the inhalation and development of secondary agents (probably streptococci), which may readily take place. After the original diphtheria has disappeared the secondary lobular pneumonia may long prevent recovery or even cause a fatal termination of the illness. The condition of the heart in diphtheria is of especial importance, because severe functional impairment, probably of toxic origin, is common. Even when the fever is slight the pulse may sometimes be very rapid, small, and often irregular. An abnormal slowness of beat is much rarer. It is especially to be borne in mind that even in apparently mild cases there may be very dangerous cardiac weakness. The latter depends in part on degeneration of the heart muscle fibers, in part on microscopic focal areas of acute interstitial myocarditis. The clinical symptoms of this acute infectious myocarditis (Romberg) are arrhythmia, inequality, acceleration (more rarely retardation), and weakness of the pulse. There is often cardiac dilatation, and as an indication of beginning disturbance of circulation, hepatic enlargement and a diminished amount of urine. Sometimes death occurs suddenly and unexpectedly from cardiac paralysis. In other cases the manifestations of cardiac weakness last several days or even weeks, either to end fatally or to clear up slowly. The blood often shows a distinct leucocytosis.

Since nephritis is especially common in septic diphtheria, it is hard to decide whether it is due to the primary disease or is to be considered a secondary complication. Its clinical significance is not very great, since it neither materially modifies the general picture nor has any definite bearing on the result of the disease. Œdema, uræmia, etc., are only rarely seen in diphtheritic nephritis. The macroscopic changes in the kidney are usually very slight. Microscopically we find the various degenerative conditions of acute nephritis.

The Nervous Sequelæ of Diphtheria.—The convalescent from diphtheria is liable to be attacked by certain nervous sequelæ. Of these, diphtheritic paralysis is the most important. It appears about one or two weeks after the throat trouble ceases, or perhaps earlier, and it is quite as likely to follow mild cases as severe ones. It attacks the soft palate by preference. The voice becomes nasal and deglutition difficult. The nasopharynx is imperfectly cut off during the act of swallowing, and with each attempt liquid regurgitates through the nose. Usually the pharyngeal mucous membrane is hypoæsthetic at the same time, and deprived of its reflex excitability. There may also be paralysis of the vocal cords upon one or both sides, and this again is frequently combined with anæsthesia of the mucous membrane of the throat. There may be paralysis of the ocular muscles, of which those controlling accommodation are most apt to be affected, rendering the vision for near objects imperfect. Paralysis of the muscles of the trunk and extremities is least frequent, but it may be very extensive. Sometimes several of these parts are paralyzed simultaneously. Thus we see quite often paralysis of the soft palate and of the muscles of accommodation combined. In some cases there is well-marked ataxia of the lower limbs with or without paresis. This renders the

gait very uncertain and tottering, the tendon reflex is almost always abolished, while sensation is affected slightly if at all. Very rarely diphtheria is followed by contracture of the hands or other parts, by difficulty in articulation and paresis of the bladder. A paralysis of the pharynx is sometimes left behind, so that the children have to be fed for weeks through an œsophageal tube. It is a remarkable fact that not only in almost every case of the nervous disorders which we have mentioned, but often also in individuals who have entirely escaped them, there may be no patellar reflex after diphtheria for weeks or even months. With regard to the pathological state, it is probably a degenerative condition of the corresponding peripheral nerves, not only in the post-diphtheritic paralysis, but also in the cases of post-diphtheritic ataxia (see the chapter on primary multiple neuritis in Vol. II). These degenerations are probably due to the poisonous chemical action of the diphtheria toxins. It is of great interest to note that paralysis has also been produced experimentally in animals by the action of diphtheritic toxin by Roux and Yersin and others. The prognosis of all the nervous sequelæ of diphtheria is very favorable, and even in severe cases complete recovery usually takes place in the course of a few weeks or months. This is in harmony with the peripheral nature of the disease. Only the rarer paralyses of the respiratory muscles (diaphragm) and of the heart (*vide supra*) are really very dangerous.

Diagnosis.—Errors in the diagnosis of diphtheria may be made in two ways. In the first place, a simple lacunar and necrotic angina may not infrequently be pronounced a diphtheria, and the parents and relatives of the patient thereby unnecessarily frightened. The second error arises through a failure to properly recognize milder grades of the disease, and, in consequence, necessary precautionary measures are not taken. In very many cases the trained physician can differentiate between diphtheria and follicular tonsillitis by a purely clinical examination, above all by an examination of the throat. A positive differentiation cannot, however, be made in all cases. The diagnosis of diphtheria is almost certain if there is a true membrano-croupous exudate or patch that extends beyond the tonsils to the soft palate or the uvula. Follicular tonsillitis, on the other hand, is readily recognized where punctate follicular plugs are present. The innumerable bacteriological examinations of the past few years have alone made it possible definitely to establish the fact that true diphtheria sometimes manifests itself as a mild suppurative catarrhal or follicular inflammation. In such cases the diagnosis can only be made by the demonstration of the bacilli. If such examination be impossible, one should be on his guard, particularly if the patients are children.

The bacteriological diagnosis of diphtheria should at first be attempted with a smear stained with Löffler's alkaline methylene blue. The younger diphtheria bacilli (*vide* Figs. 12 and 13) present the characteristic club- or wedge-shaped thickenings at their ends. They are in addition often slightly bent, diverge from one another and lie irregularly crossed, and frequently they are grouped in small clumps or "nests." The older bacilli are somewhat longer, their ends are more thickened, and they often show a segmental degeneration. The diphtheria bacillus is nonmotile, and reveals no spore formation. The growth of the bacilli on Löffler's blood serum is a further means of differentiation. The growth consists of small, wax-white colonies that present a characteristic appearance under the low power of the microscope. A

definite differentiation between true diphtheria bacilli and the so-called pseudo-diphtheria bacilli is obtained by staining the bacilli from a fresh culture according to M. Neisser's method (double stain with methylene blue and Bismarck brown). With this the bacilli are colored brown and the granules at their ends blue (*vide* Fig. 13).

Fig. 14.—Diphtheria bacilli. Smear from the tonsil.

[When the membranes are confined to the nose, the diagnosis may be more or less difficult; but it is especially in cases in which the nasal mucous membrane is involved that we encounter great swelling of the glands at the angles of the jaw. There is also apt to be a thin, acrid, bloody, or sero-purulent discharge.

Jacobi states that while diffuse pharyngeal injection may or may not point to imminent diphtheria, marked local congestion is either traumatic or diphtheritic. An examination of the urine should never be neglected in doubtful cases; in diphtheria a trace of albumen is very common; in simple or follicular sore throat albumen is very rare, if indeed it occurs at all.]

Prognosis.—The dubious prognosis of true diphtheria is universally known, even by the laity. The very fact that the best-developed and healthiest children so often fall victims to it associates the name diphtheria with the saddest memories. There are indeed many mild cases which recover in a week or two, and severer ones which end happily in three or four weeks; but in most cases, where the process extends into the larynx, or the symptoms of a severe constitutional infection occur, the prognosis must be regarded as very serious. Recently, however, since the introduction of the serum treatment, the prognosis even of severe diphtheria has become considerably more favorable. Formerly, before the serum treatment, it was regarded as a relatively favorable result if one half of the children with diphtheria in the hospitals, on whom tracheotomy was done, recovered! Often sixty or seventy per cent of the children operated on died. At present, however, the mortality of the tracheotomy cases, treated at the same time with serum, has fallen to about twenty-five per cent, and these are only the severe cases in which the serum treatment was begun late. The improved prognosis under the serum treatment is much more favorable if we compare the whole number of cases and the death-rate of diphtheria before and after the introduction of this treatment (Heubner, Widerhofer, Baginsky, etc.).

In general, diphtheria is to be regarded as the more serious the younger the child is. In later childhood, after the eighth or tenth year, the number of dangerous cases is much less. The description of the symptoms shows sufficiently what the dangers of the disease are, and how they are to be recognized. We would state once more that, even in apparently mild cases, the danger of sudden paralysis of the heart, though fortunately rare, always demands the greatest caution.

Treatment.—Up to a [comparatively] short time ago it was impossible to say anything in regard to any generally recognized treatment of diphtheria which was really effective; but in 1893 a method of treatment was discovered, chiefly by the important and interesting investigations of Behring and

Roux, which deserves to be called an actual specific. Further observations have established this treatment as one of the most brilliant and triumphant medical acquisitions.

Behring found, in continuing the important discoveries of Pasteur, Buchner, and others, that certain protective matter ("antitoxin") was formed in the blood serum of those animals (sheep, goats, horses, etc.) which had been infected with artificially weakened cultures of diphtheria bacilli. If the animal survive the milder infection, it is less sensitive to a severer infection, and, finally, by continued inoculation, it may even become immune to the severest infection. The blood serum of such immune animals (horses are now almost invariably employed at the serum establishments) can now be used in the treatment of diphtheria in man. In order to have a definite measurement for the toxins and antitoxins, the term *toxin unit* is employed to indicate the smallest amount of poison which, in four days, will kill a guinea pig weighing 250 grams; while the term *antitoxin unit,* or the unit of immunity ("Immunitätseinheit," "I. E."), represents the amount of serum which will prevent the action of 100 toxin units. Certain theoretical objections have, however, been raised against this method of measurement, but it is still in practical use. A "simple serum" is one in each cubic centimeter of which there is the equivalent of one antitoxin unit. Thus, for example, in a bottle of "Höchst Serum" containing 5 c.c. of a "two hundredfold serum," there is contained the equivalent of 1,000 antitoxin units.

The treatment is simple, and consists in injecting the serum under the skin of the child who has diphtheria by means of a carefully disinfected syringe. The best places for injection are the anterior chest wall or the thigh. Since the amount of serum to be injected is usually about 5 c.c., we should use a special syringe which is not too small. The best are made like a Pravaz syringe with an asbestos packing. It is to be understood that the injections must be made with the strictest aseptic precautions. The syringe and needle are sterilized by boiling, and the site of the injection is carefully cleansed with alcohol and ether. There are different preparations of serum of equal reliability on the market. We ourselves have almost exclusively employed that made by the Höchst Anilin Dye Works. It is put up in three strengths: the curative dose of No. I equals 600 antitoxin units, that of No. II 1,000, and of No. III 1,500 units. In addition, a "concentrated serum," of dosage up to 3,000 units, has recently been manufactured.

In early and milder cases we inject Serum I ("600 units of antitoxin"), in severer and more advanced cases, especially if there be any signs of disease in the larynx, we use at once Serum II ("1,000 units of antitoxin"), or, even better, Serum III. According to the severity of the case, or according to its course, we repeat the same injection or a weaker dose of the same once in the next twelve to twenty-four hours. In many cases of moderate severity a single injection of Serum II suffices. In adults, one usually begins at once with Serum III, or, if the case is severe, the "concentrated serum," in doses of 2,000 to 3,000 units, may be given.

[These doses of antitoxin are smaller than is the custom in America: for a child of one year, an initial dose of 2,000 or 3,000 units, and in laryngeal cases 3,000 to 5,000 units; for an adult 4,000 to 6,000 units. If treatment has been delayed, or if the case is severe, larger doses are required. J. H.

McCollom has emphasized the efficacy and the harmlessness of large doses and the need of repeating them until local and general improvement is evident. The danger lies not in using too much but in using too little.]

The favorable action of the serum is shown chiefly by the fact that the affection does not extend after the injection. The existing croupous deposit is thrown off in the course of the next four or five days.

The serum injections have no injurious action. Urticaria sometimes occurs after an injection, but it has no serious significance. Taking everything into consideration, it is therefore the duty of the physician, in accordance with our present knowledge, to use the serum in every case of true diphtheria in children. In suspicious cases, too, it is wiser to give an injection at once, without waiting for the substantiation of the bacteriological examination. It is unquestionable that deaths from diphtheria occur even with serum treatment. These are either cases in which treatment has been begun too late, or cases in which there is a septic mixed infection. The fact cannot be denied that the results in some severe epidemics were not as good as were obtained at other times. Nevertheless, the vast majority of observations to the present time speak emphatically in favor of the Behring method. If we survey the discoveries made in the field of serum studies as well as in its related branches, we may truly hope that we physicians are standing on the threshold of an entirely new era in the treatment of infectious diseases.

It is of the greatest practical importance that the serum treatment be begun as early as possible. The most favorable results are seen in children treated on the very first days of the disease. In these cases the development of post-diphtheritic paralysis appears to be much less commonly seen. Local treatment of the affected places in the pharynx at the same time is unnecessary. We would, however, emphasize the importance of a general cleansing and disinfection of the whole oral cavity by gargling and rinsing with dilute solutions of potassium chlorate, hydrogen peroxid, boric acid, and the like. Ice bags or hot or Priessnitz compresses may be applied externally. Internal medication (potassium chlorate, potassium iodid, etc.) is entirely unnecessary. It is also very important to watch the child's nutrition carefully in order to keep up the strength. Milk, eggs, meat juice, small quantities of wine, etc., are most advisable.

If the larynx is attacked, and if, in spite of the serum treatment, the consequent laryngeal stenosis threatens to cause suffocation, tracheotomy is our only resort. It is never indicated by the disease itself nor by the severity of the case, but only by persistent obstruction of the larynx. It is therefore not invariably easy to decide whether tracheotomy is called for in any particular case. If the general condition be bad and respiration already impaired, it is often very difficult to determine whether laryngeal stenosis exists. Tracheotomy will be of no avail if the croup has already extended to the bronchi, or if the dangerous condition of the patient is due to the severity of the constitutional infection or to incipient paralysis of the heart. We have already said that, since the introduction of the serum treatment, the prognosis of diphtheria has become better even in the tracheotomy cases. How tracheotomy is performed, and in what the after-treatment consists, must be learned in the text-books on surgery, where we will also find a description of the so-called intubation of the larynx.

If severe pulmonary symptoms occur in diphtheria, we should use first of all cool or hot packs of the whole body or tepid baths with cool effusions. Such cutaneous stimulation refreshes and enlivens the whole nervous system, and we should therefore try it also in the severe cases with secondary septic infection. In septic diphtheria we must also pay especial attention to the condition of the heart. So far as is possible we should try to avert the threatening cardiac paralysis by stimulants, such as wine, camphor, and strophanthus.

The greatest precautions must be taken in all cases of diphtheria in which even the slightest signs of cardiac weakness and arrhythmia appear. The children must be kept perfectly quiet in bed as long as possible, and must be most carefully nourished and looked after.

The nervous sequelæ of diphtheria are best treated with the constant current. As an internal remedy, iron is good, and also nux vomica or strychnin. The last may be given subcutaneously, if desired, in doses of $\frac{1}{60}$ to $\frac{1}{30}$ gr. (gm. 0.001 to 0.002).

The prophylaxis of diphtheria demands that every child with diphtheria be absolutely isolated from healthy children. The isolation is not to be terminated too early, since virulent bacilli may remain in the mouth and throat even weeks after recovery. The sick-room, as well as everything that has come in contact with the patient (bedding, toys, etc.), must be carefully disinfected. According to Behring, a healthy child may be positively protected from infection by diphtheria for two or three weeks by the injection of a small dose of serum—the whole or one half of Dose I—that is, about three hundred units of antitoxin. It is, of course, very hard to pass final judgment on this, and for the present, therefore, it must be left to the discretion of the physician in what cases he will try prophylactic injections of serum.

[Diphtheria is a disease which involves commonly much exhaustion, and too much stress can hardly be laid on the importance of administering the maximum amount of nourishment in the most assimilable and easily swallowed forms from the start.

It is also important to give stimulants early in most cases, not waiting for signs of exhaustion. Enormous quantities of brandy can often be given to small children without the slightest toxic effect. No general rule can be laid down; the requirements of each case must be studied and met.

When painful deglutition interferes with nutrition, peptonized milk, eggs, brandy, and the like, must be given by the rectum. Rectal alimentation and stimulation and feeding with the œsophageal tube are also to be resorted to in cases of post-diphtheritic paralysis of the œsophagus.]

CHAPTER XI

INFLUENZA

(La Grippe)

Influenza is a specific, acute, infectious disease which is especially distinguished by the occasional enormous extent of its epidemics. While often years

and decades pass without any especial attention being called to the disease, suddenly cases of it will appear with such frequency that the largest part of the population is attacked, and the disease may better be described as pandemic than epidemic. Pandemics of influenza can be traced back with certainty into the sixteenth century. In the present century the influenza during the years 1830–33 traversed almost all of Asia and of Europe, then later there appeared numerous smaller epidemics, but these aroused general attention so little that the disease, upon its last pandemic appearance in the winter of 1889–90, was almost unknown to many physicians. Since that time the disease seems never to have wholly disappeared, as a few cases, and occasionally small groups of cases, are constantly seen.

Ætiology.—The organized agent of influenza is, in all probability, the "influenza bacillus" discovered by R. Pfeiffer. It is invariably found in the bronchial secretion of patients with the "catarrhal form" of influenza (*vide infra*), a definite form of very small bacilli with rounded ends which stain darker than the central portion. These bacilli either are free in the mucus or (especially in the later stages) are found in the pus cells. They grow best on plates of agar smeared with blood. The influenza bacilli very soon die if dried, and also if put in water.

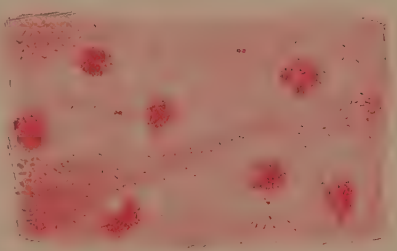


Fig. 15.—Influenza bacilli in the sputum of a case of broncho-pneumonia.

In all probability the infection is usually due to inhaling the germs. The bacilli seem to appear at certain times over an immense territory, so that they are scattered everywhere through a large extent of country. Many observations upon the appearance of the disease in isolated institutions (convents, and the like) render it very probable that the poison may also be carried by a person suffering from the influenza to another in regions previously unaffected. Nevertheless, this contagious manner of spreading plays no great rôle in comparison with the indirect spread of the disease, this latter mode being everywhere possible during an epidemic of influenza by inhalation of the bacteria.

There is scarcely any reason for speaking of especial predisposing causes of influenza, inasmuch as at the time of a well-marked epidemic the overwhelming majority of the population are attacked, both the healthy and the diseased, the vigorous and the feeble. Sex certainly makes no difference, and age only to this extent, that the disease is seen more rarely in little children under one year of age than in older children and adults. That catching cold has no special ætiological significance is evident from the fact that influenza often appears in patients who are already sick in bed.

It should finally be mentioned that animals also, and, in particular, horses, may be attacked by the influenza; but, nevertheless, it is as yet a doubtful question whether all the diseases in animals which are described under this name are actually identical with genuine influenza.

Symptoms and Clinical History.—The best general idea of the extremely manifold symptoms of the disease will be obtained if we bear in mind that the influenza causes both a marked infectious (or toxic) general constitutional

disturbance of the body, and also certain local lesions with local symptoms. The clinical picture therefore varies greatly according to the predominance of one or the other group of symptoms, and also according to the special form of the local disease.

The onset of influenza is generally rather sudden. As a rule, the marked cases begin with rather high fever, ushered in with a chill, violent headache, marked constitutional depression, and usually considerable pain in the back and loins. The weakness of the patient may be so great that, even if a vigorous individual, he will at once take to his bed. Severe nervous symptoms, such as stupor and delirium, are exceptional. Sometimes, but not very often, there is initial vomiting. The backache is often associated with pains in the muscles and joints. Oppressive pain in the eyes also is quite characteristic. This is particularly felt upon moving the eyeballs, and therefore is probably located in the external muscles. The spleen is occasionally somewhat swollen, but any great increase in its size is exceptional.

If the clinical symptoms as the case progresses are mainly limited to the above-named constitutional symptoms—fever, languor, headache, pain in the muscles—we may speak of a "typhoidal form" of the disease. Usually, however, certain local symptoms put in an early appearance, and it is especially the respiratory apparatus which is attacked. The precise symptoms vary considerably in different cases. Sometimes the upper portion of the respiratory tract, the nose, larynx, and trachea are involved; sometimes, from the start, the smaller bronchi. In the first instance there is marked coryza, not infrequently accompanied by conjunctivitis; in the other case there is cough, due to a dry bronchitis, which can be easily detected upon auscultation, and which involves especially the lower portion of the lungs. If these local symptoms outweigh the constitutional, the case is described as belonging to the "catarrhal form" of influenza.

Sometimes the influenza is localized in the digestive apparatus. This "gastro-intestinal form" is much rarer than the catarrhal. In this case, in addition to the more or less strongly characterized constitutional symptoms, there is marked disturbance of the stomach and intestines, as shown particularly by nausea with persistent vomiting, diarrhea, abdominal pain, etc. In one case we observed jaundice. The frequent reddening of the anterior gums and the swelling of the papillæ at the tip of the tongue (Franke) should also be noticed.

The pains in the back, loins, and extremities already mentioned may persist with unusual violence, and this peculiar form of the disease is known as the "rheumatoid." The muscles and the muscular attachments are probably the chief seat of these pains, which may be so violent that the patient is unable to lie comfortably, and sometimes keeps up a continual moaning. The loins, in particular, may be the seat of most acute pain, also the upper arms, the knees, the thighs, and the eyes. Objective changes in the painful parts, such as swelling of the joints, are scarcely ever seen, nor are the nerve-trunks, as a rule, especially sensitive to pressure. The painful muscles are usually weaker than normal.

The grouping of the clinical varieties of influenza under the four forms already named affords a general idea of the manifold symptoms of the disease, but this division into separate forms must not be carried out too strenuously,

for in reality many cases of the disease occur which present transition forms and combinations of the various groups of symptoms. Moreover, in all the forms a distinction must be made between mild and severe attacks, for in influenza, just as in most other infectious diseases, there are many rudimentary and mild cases as well as the fully developed ones, and some could not be properly interpreted but for the presence of the epidemic.

The duration of the disease is best determined by the duration of the fever. In the very mildest cases there may be no fever whatever, or simply a slight evening rise of temperature. As a rule, there is a moderate fever, between 101.5° and 103° F. (38.5° and 39.5° C.), although higher temperatures even to 104° F. (40° C.) and more are not infrequent. In the beginning of every severe attack the fever rises abruptly. After a duration of several days (four to seven) it may fall again in a manner approaching a crisis. More frequently, especially when there exists diffuse, catarrhal trouble in the lungs, the fever ends by lysis. With comparative frequency there are found to be noticeable deviations in the temperature curve (see Fig. 16); thus, for example, the high fever of the onset sinks on the second or third day, to be followed by an almost afebrile period of one or two days, whereupon a marked rise of temperature ensues. With this change in the temperature there are usually also corresponding variations in other symptoms.

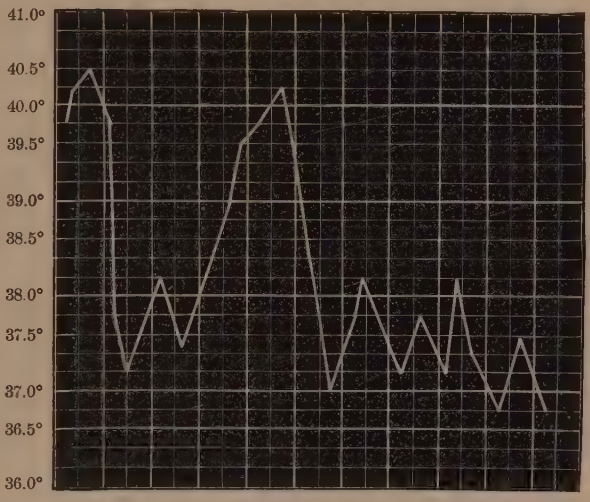


Fig. 16.—Example of a double-pointed fever curve in influenza. The second elevation indicates the onset of a mild catarrhal pneumonia. (Erlangen Medical Clinic.)

The heart, in influenza, is frequently involved to a marked degree. The pulse is usually proportionately rapid. In individuals whose heart muscle has previously been weakened, and especially in the aged, great cardiac weakness (arrhythmia, dilatation), even endangering life, may occur.

The duration of simple, uncomplicated influenza is in the mild cases about three or four days, in the severer cases about seven to ten days. To be sure, we should also consider in this connection that convalescence is often surprisingly slow, so that the after-pains (as it were) of the disease are felt for weeks. These consist, for instance, in a certain degree of debility, and in myalgia. Sometimes also there are complete relapses, so that directly or a short time after the disease has ended the symptoms begin anew. The special form of the disease may change in this case, so that, for example, the relapse of an influenza with predominant constitutional symptoms assumes the pronounced catarrhal form. Again, it is not very rare for a patient to

suffer from two or even more attacks of influenza separated by a considerable interval of time.

Complications and Sequelæ.—While all the symptoms of influenza which we have thus far described are the direct effects of the original pathogenic cause, the majority of the frequent complications are undoubtedly dependent upon the ingress of secondary infection. The system when attacked by influenza is greatly exposed to these secondary influences, and almost all the dangerous and tedious cases of influenza become such only because of a mixed infection of this sort. This is particularly true of the lungs, in which secondary disease occurs most frequently—sometimes even in the first days of illness, but also in other cases later. The conditions here are similar to those seen in measles and whooping cough. The simple, mild catarrh belongs to the original disease; the severe pulmonary affections are, however, invariably secondary complications occasioned by new pathogenic influences. These influences are not always the same. According to the investigations of Ribbert, Finkler, and others it is chiefly the pneumonia diplococcus and the streptococcus which are the true excitants of the secondary pneumonia seen in influenza. These cases of pneumonia are either extensive catarrhal pneumonia especially affecting the lower lobes, or more rarely croupous pneumonia with its characteristic sputum. We must also add that the influenza bacilli themselves may excite a lobular pneumonia which shows a marked purulent character.

Quite often the pneumonia results in localized suppuration in the lungs. Influenza pneumonia is not infrequently seen as a relapse in cases apparently recovered from influenza. The temperature again rises, and with it dangerous cardiac and general weakness is often combined.

If pneumonia be added to influenza, the former overshadows the whole picture. The patient is oppressed for breath, has a severe cough with profuse expectoration, looks pale or cyanotic, and suffers from high fever. These symptoms persist for two or three weeks, and then gradually abate. It is in this way that influenza becomes dangerous for elderly and feeble or sickly persons. Some influenza pneumonias have a more prolonged course, lasting from four to six weeks and even longer. Such cases usually end in recovery. It is only exceptionally that the pulmonary disease leads to atrophy and bronchiectasis, pulmonary abscess, and similar conditions.

Comparatively often pleurisy with effusion is conjoined with the influenza pneumonia. The exudation is generally serous, but exceptionally it is purulent.

Of the other complications, the frequent occurrence of inflammation of the middle ear should be especially mentioned. Serious affections of the eye (keratitis) are much more rarely seen. We have several times observed acute nephritis, but this has always pursued a mild course. Among cutaneous eruptions, herpes labialis is a frequent phenomenon in all forms of influenza, even the milder. Other exanthems, such as urticaria and roseola, are much less frequent.

Many of the complications named may continue even after the fever and all other symptoms have ceased, so that they must be regarded as sequelæ. This is particularly true of the diseases of the ear and eye and of persistent bronchitis, of pneumonia assuming a chronic form, etc. An important and, for the patient, a troublesome and painful sequel is furunculosis, especially if some

of the boils are located in the axilla or near the anus. Very often neuralgic pains in the distribution of the trigeminus or in the course of the sciatic or other nerves will persist for a considerable time after the influenza has ceased.

Painful disturbances in the muscles, periosteum, or in the bones may persist for a long time. In a few cases more severe nervous sequelæ, such as spinal symptoms, psychoses, and neurasthenia have been seen after influenza. A special form of acute hemorrhagic encephalitis (see Vol. II) stands in relationship to influenza. Finally, permanent disturbance of the heart's action (cardiac weakness, instability, and irregular pulse) is sometimes observed after influenza. Franke groups all these often tedious sequelæ under the term "chronic influenza." This diagnostic term should not, however, be abused.

Diagnosis.—The diagnosis of influenza is in general not difficult if one has to deal with a well-marked case at the time of an influenza epidemic. The characteristic initial symptoms of fever, headache, and pain in the loins are to be considered first of all. Their onset is much more rapid than, for example, in typhoid fever. Later on the pain in the various muscles as well as the catarrhal symptoms are the most characteristic phenomena.

Uncertainties and errors in diagnosis may arise from the fact that, on the one hand, we are disposed at the time of an epidemic to call almost all the catarrhal and indefinite mild affections we see "influenza," while, on the other hand, when there is no epidemic, we cannot positively distinguish the sporadic "cases like influenza" from the ordinary acute febrile bronchitis, etc. It is indeed very possible, *a priori,* that different morbid agents may produce similar mild acute diseases of the accessible mucous membranes. In such cases, with pronounced general symptoms, high fever, etc., we must often be content with the diagnosis of "infectious" laryngitis, bronchitis, etc., without being able to decide definitely whether the case is to be classed ætiologically with influenza or not. At any rate in practice we should not abuse the diagnosis of influenza. An absolute diagnosis can only be made by bacteriological examination, although this, as yet, is often quite a difficult matter.

Prognosis.—For an individual who is healthy and vigorous, influenza is not a dangerous disease, even in its severer forms; for elderly persons or invalids it may, however, be a serious affection. Patients with heart disease or pulmonary disease, or those suffering from chronic nervous troubles, sometimes succumb to it; so that the general mortality at the time of a great epidemic of influenza is always considerably increased. The above-enumerated complications are by far the most dangerous; less often is a fatal termination caused by general or cardiac weakness. The above-mentioned sequelæ are also to be considered in prognosis.

Treatment.—No specific remedy for the disease is known. In general we must, therefore, pursue a purely symptomatic method of treatment. For the initiatory fever, the headache, and the pain in the loins, antipyrin is sometimes a good remedy, and the same may be said also of phenacetin, aspirin, and especially salipyrin. These drugs as well as soothing liniments are also prescribed for the persistent pains in the muscles. In many cases presenting marked catarrhal manifestations diaphoretic treatment acts well. The treatment of the pulmonary complications is according to the established methods. Morphin, codein, Dover's powder, and similar drugs are used for the troublesome cough. Apomorphin, senega, and other expectorants may be employed,

and, if indicated, external remedies such as an ice bag, moist applications, or dry cupping. Stimulants (champagne, camphor, caffein, strophanthus) are indicated for beginning cardiac weakness. Inasmuch as influenza, as already stated, is not infrequently followed by a protracted state of general weakness, the patient must be urged to take due care of himself during the entire period of convalescence.

CHAPTER XII

DYSENTERY

Ætiology.—By "dysentery" is meant a disease of the colon, which appears sporadically, but more often in epidemics; it is excited by infection with an organized pathogenic poison, about which we have as yet no definite knowledge; and the infection is probably at first a local one. From the investigations of various men, especially Kartulis, it seems very probable that a certain form of amœba must be regarded as the cause of "endemic tropical dysentery." At least in endemic dysentery we almost invariably find, both in the stools and in the walls of the dysenteric intestinal ulcers and in the dysenteric abscesses of the liver, many amœbæ (amœba coli), which are never found in any other intestinal affection. Dysentery may also be artificially produced in cats by means of feces containing amœbæ. In dysentery as it occurs in Germany, however, we do not apparently find amœbæ in the stools, as a rule, although even in Germany Quincke and others have described certain cases of "acute enteritis" ("amœbic enteritis") in which there were a large number of amœbæ present. Most of the epidemics in Germany have been caused by the Bacillus dysenteriæ, described by Kruse, Shiga, and others. This bacillus resembles the typhoid bacillus, but is shorter and thicker, has no flagella, and is therefore nonmotile.

Fig. 17.—*Amœba dysenteriæ* (after Roos). *a*. Amœbæ without foreign bodies. *b*. Amœba containing blood. *c*. Amœbæ showing vacuoles. *d*. Young forms. *e*. Encysted forms.

The true home of dysentery is in warmer and tropical countries, where the disease is much more violent and widespread than here. For example, the mortality among the soldiers of the Anglo-Indian army due to dysentery is said to be at times thirty per cent of the entire number of deaths. In our climate most of the epidemics occur at the end of summer and in autumn. Endemic influences are certainly important. The special conditions in some places are evidently very favorable for the development and dissemination of dysenteric germs, and there are

other places equally unfavorable. There can be no other explanation of the immunity of some localities contrasting with the great prevalence of the disease in others. How infection occurs we do not yet know. Many observations, especially in southern countries, support the idea that the germs may be taken into the system by drinking water. It is very probable that in addition, dysentery can be spread through the medium of the fecal dejections of the sick—e. g., from privies, chamber vessels, and bed linen. Many cases were formerly referred to catching cold or to some error in diet; but we must, of course, regard these merely as predisposing influences.

Pathology.—The objective pathological lesion of the colon, in all severe cases, consists in a pronounced croupous-diphtheritic inflammation. The remarks as to the general pathology of such inflammations made in the section on pharyngeal and laryngeal croup (p. 77) are equally applicable to the analogous dysenteric inflammation. In this case, too, there is first a destruction of the epithelium and then the formation of a fibrinous exudation occupying its place, and penetrating down into the tissue of the mucous membrane itself. At the same time there is an intense purulent infiltration of the mucous and submucous tissue, accompanied by extensive ecchymoses. In the most virulent cases the macroscopic appearances are marked thickening of the whole wall of the intestine, congestion of the serous layer, and the conversion of the inner surface into a mottled, dark-red, irregularly roughened area of ulceration. The disease may be confined to the rectum and the sigmoid flexure, but in severer cases it involves the entire colon as far as the ileocæcal valve, or even extends to the lower portion of the ileum. Besides this severe form of diphtheritic or even gangrenous dysentery, there is a milder variety, termed catarrhal dysentery. In this the mucous membrane is found in a state of intense purulent inflammation, with ecchymoses. Even here little masses of croupous exudation, which can be torn off, have replaced the epithelium; but they never form continuous layers of great extent. There is no sharp boundary line between the two forms, the milder catarrhal-croupous and the severer diphtheritic dysentery. Numerous transitional and combined varieties exist.

We must remark, in conclusion, that precisely the same anatomical changes as are presented in true dysentery may result from other causes. Important among these is persistent fecal impaction in the rectum, which, by a purely mechanical effect upon the epithelium, may excite a diphtheritic inflammation in the mucous membrane. And any severe constitutional disease whatsoever, such as typhoid fever, measles, smallpox, septicæmia, or phthisis, may be attended by a so-called "secondary dysentery." This is most frequent in hospitals. Whether it has the same ætiology as genuine dysentery is uncertain.

Clinical History.—Throughout the entire illness the most prominent symptoms are intestinal. There may be first of all some slight irregularity of the bowels for a few days, and then appears a moderate diarrhea. The stools are at first feculent, although thin, and number two to six daily. After a few days the discharges increase in frequency, and become extremely characteristic.

The stools are very frequent, occurring ten to twenty, and even sixty or more, times, in twenty-four hours. In severe cases there may be a distressing and almost constant desire to evacuate the bowels. After every operation, and to some extent during it, there is tenesmus attended by intense burning pain

in the anus. The stools soon lose their usual feculent character in great part if not entirely. They become scanty, so that not more than about half an ounce is evacuated each time. For the most part they usually consist of a sero-mucous fluid, in which are suspended numerous shreds and particles of varying size. These are blood-stained bits of mucus, little coagula of blood, and necrosed pieces of mucous membrane. One or another of these constituent parts may predominate, so that there may be slimy, purulent, or bloody stools, or all sorts of combinations of these varieties. We often find, besides, a few small masses of feces, usually covered with mucus. We sometimes see numerous clumps of mucus, resembling sago or frog's spawn; they are probably mucous casts of the follicles. Under the microscope the greater part of the dysenteric discharge is seen to consist of pus corpuscles and blood. There are also cylinder epithelium and an enormous amount of detritus, and the bacteria of putrefaction. A purely dysenteric stool has no bad odor, except that in the worst cases of gangrenous dysentery the discharges become blackish and extremely offensive.

Besides the rectal tenesmus there may be a cramp-like pain during micturition. There are often violent attacks of colic. The abdomen is usually rather tense, and tender on pressure along the line of the colon, but without tympanites. The anus may be red, inflamed, and excoriated. Gastric symptoms are on the whole infrequent, if we except the complete anorexia which exists in all severe cases. Sometimes there is repeated vomiting. Occasionally hiccoughs prove distressing. The tongue usually has a dry, greasy coating.

The symptoms just depicted last about a week or ten days. If the case is of much intensity, the general condition is also greatly affected. The patient seems much collapsed, and is very languid and feeble, with a small and rapid pulse. The skin becomes cool and rough, the voice weak and hoarse. There is pain in the muscles. The patient wastes away. The temperature has little that is characteristic or typical. In many cases there is no fever at all, and the temperature may even be subnormal. In most cases, however, there is an irregular fever seldom exceeding 104° F. (40° C.), and having remissions.

In the worst cases the general weakness may increase more and more, and death occur; but with us a favorable termination is much more frequent. The distress gradually diminishes, the stools assume more and more of a feculent character, the patient becomes stronger, and after one and a half to three weeks convalescence is established. It may be a long while, however, before a patient completely recovers from a severe attack. A third possibility is the transition of the acute into a chronic dysentery. In this the symptoms of a chronic colitis, usually attended with cachexia, may persist for months and years.

Mild, rudimentary forms of dysentery also occur, presenting no severe intestinal symptoms, and recovering at the end of a few days. In these cases, too, great sensitiveness of the intestine to disturbing influences frequently persists for quite a long time after the illness. There may be exacerbations of the disease, and relapses.

Complications.—Complications of dysentery, localized in other organs, are rare, at least in epidemics here. In tropical dysentery abscess of the liver is comparatively common. It is dependent upon metastatic processes from the portal radicals. In some cases abscesses of the lungs and of the brain may

follow abscess of the liver. Inflammation of the serous membranes or of one or more joints may also occur. If paraplegia follows it may be referred either to secondary myelitis or to polyneuritis. Dysentery is also said to occur in connection with a "general scorbutic diathesis," but to all appearances this is usually a "septic" complication. The dysenteric ulcers rarely lead to perforation and consequent peritonitis.

Diagnosis.—The clinical diagnosis is seldom very difficult. It is based exclusively upon the intestinal symptoms and the character of the stools. It is only the cases of secondary dysentery which occur in the course of other severe diseases that are likely to escape observation.

The bacteriological demonstration of the dysentery bacilli in the stools may be made by means of the Drigalski-Conradi nutritive medium. The bacilli grow in colonies of a bluish color, as do typhoid bacilli, but the dysentery bacilli are nonmotile, and are agglutinated by the serum from dysentery cases. The amœbæ of dysentery are detected by microscopical examinations of the stools.

Prognosis.—The prognosis is mainly influenced by the character of the epidemic, which, as we have said, is in our climate usually benign. There may be danger, particularly to elderly people, from bodily weakness and collapse.

Treatment.—Prophylaxis demands that the isolation of the patient and the disinfection of the stools be as complete as possible. The healthy must be very careful during an epidemic not to catch cold, and to avoid errors in diet, for experience shows that an opposite course predisposes to the disease.

The patient must be kept warm, and must not leave his bed, even if the attack be mild. The diet must be rigorous. If the strength is fair, thin porridge, milk, and broths suffice for some days. To a feebler person we should give somewhat stronger nourishment from the start, e. g., eggs, peptonized meat, and wine. Most patients bear liquids that are lukewarm better than those which are cold.

As to drugs, the habit of almost all experienced physicians is to give at first a mild laxative. Although opium does not usually control the diarrhea and tenesmus at all, it is the rule for decided improvement to follow the exhibition of the laxative. During the first days, or, if need be, later, we give two to four tablespoonfuls of castor oil daily. If this medicine is very disagreeable to the patient, we can replace it by a strong infusion of rhubarb (10-to-100). In southern countries large doses of calomel (gr. viij to xv, gm. 0.5 to 1) are customary, and are highly praised by the physicians there. Further on in the disease we may content ourselves with giving mistura amygdalæ; or we may administer bismuth in the following mixture: Bismuthi subnit. vel salicylat., ʒj (gm. 5); mucilaginois acaciæ, syrupi simpl., āā ʒss. (gm. 15); aquæ destil., 120—to be shaken before taking. If the disease should get worse again, however, we should always try a laxative.

Emetics at the beginning of the disease are often employed in the tropics, but seldom with us. Ipecacuanha (*radix antidysenterica*), given in large doses of 15 to 30 gr. (gm. 1 to 2) is even regarded by many as a specific. Among antiparasitic remedies, naphthalin (gr. viij, gm. 0.5, thrice daily) and salol (1 to 2 drachms, gm. 4.0 to 8.0, a day) have been especially recommended. Numerous attempts have been made at local treatment by enemata. No brilliant results, however, can be claimed for any of these methods or

medicines. A decided palliative effect can be obtained from the injection of thin starch to which 20 or 30 drops of laudanum have been added. Suppositories of cocoa butter containing extract of opium often mitigate the tenesmus. Other injections are recommended, each to measure ʒij to ivss. (gm. 60 to 100), and to contain either argenti nitrat., gr. j to vj (gm. 0.05 to 0.30), or plumbi acetat., gr. ij to viij (gm. 0.1 to 0.5), or potassii chlorat., gr. xv to xx (gm. 1 to 1.5), and especially tannin (three injections a day of a warm 0.5-per-cent solution). In severe cases we may also try high injections of solutions of tannin as in cholera (*vide infra*). Many other solutions are used. The success of this treatment is, however, dubious. In all cases the margins of the anus must be protected from inflammation by frequently washing and anointing the skin.

The treatment of weakness and collapse is by the usual stimulants—wine, ether, camphor, and the like. In chronic dysentery the main point is to persevere in a strict control of the diet. We may exhibit astringents, such as tannin, tannigen, and columbo. Subnitrate of bismuth is also given, and nitrate of silver and acetate of lead. And in these chronic cases a long-continued and thorough use of rectal irrigation with fluids containing some mild astringent or disinfectant may have a good effect.

[Sporadic dysentery is a self-limited disease, and, as has been shown by Flint, runs its course within ten days without medication. Treatment, however, adds to the comfort of the patient and shortens the course. It is not customary with us to use daily laxatives. If there is any doubt as to whether the intestines have been emptied, a saline should be given, the action of which should be followed by opium in sufficient doses to allay pain and tenesmus. Subsequent action of the bowels is best obtained by simple large enemata. In weak persons castor oil is to be preferred to salines.

In epidemic dysentery active treatment is much more important. Laxatives are contra-indicated by sero-sanguinolent dejections or by asthenia, but enemata can be freely used. Stimulation is often required; nutrition must be carefully looked after, such articles being chosen as are digested and absorbed by the upper portions of the intestinal tract, leaving as little residue as possible to pass on to the inflamed colon. Opium is often demanded and tolerated in large doses, and astringents, such as the acetate of lead, gallic acid, and the pernitrate of iron, are of service.

In acute dysentery the patient should be instructed not to yield to the desire to go to stool if he can help it, and tenesmus can often be much diminished by simple irrigation of the lower bowel with water, which may be warm or cold, whichever the patient finds more agreeable.

In chronic dysentery medicated injections large enough to reach the colon are the best remedy. In amœbic dysentery, one or two quarts of a solution of quinin in water, 1 to 5,000 gradually strengthened up to 1 to 1,000, may be given twice a day; and in bacillary dysentery, three to six pints of a solution of nitrate of silver containing 12 to 60 gr. to the quart, once a day. Preliminary use of cocain may be required to lessen the sensitiveness of the rectum.]

CHAPTER XIII

CHOLERA

(Asiatic Cholera)

Historical Remarks.—The home of genuine Asiatic cholera is India. The first epidemic in that country with which we are accurately acquainted occurred in 1817. This was very widespread. The disease was probably endemic there at an earlier period. In the next few years the cholera extended in all directions, and reached Astrakhan by way of Persia. Between 1830 and 1832 the disease made its first great epidemic progress over Europe. Invading all European Russia, it reached Germany in 1831, and France and England in 1832. Then came many smaller epidemics up to 1838, when there was a complete cessation till 1846, in which year the disease, again starting from Asia, overspread Europe. There have in later years been epidemics in many places, but we cannot here enter into the particulars of them. During the war of 1866 there were many cases of cholera in Germany, and from 1883 to 1886 Italy, France, and Spain were visited by the disease. In August, 1892, cholera broke out suddenly and very unexpectedly in Hamburg, where within three months about 18,000 persons were attacked by the disease, and over 7,600 died of it. [It has not gained a foothold in the United States since 1873.]

Ætiology.—Some time ago it had become evident that the real cause of cholera consists in the infection of the system by a specific microörganism. Koch was, however, the first to succeed in the search for the poisonous agent. He was in charge of the scientific expedition sent out by the German Government in 1883 to Egypt and India for the purpose of investigating the disease. Koch found in the intestines of all the victims of cholera whose bodies he examined a certain kind of microörganism which he named the comma bacillus. At present it is often termed the spirillum or the vibrio of cholera. It is shorter than the bacillus of tuberculosis, but somewhat thicker, and it is usually bent in the shape of a comma, or even like a semicircle (see Fig. 18). In pure cultures the comma bacilli grow into long spiral threads, resembling the spirilla of recurrent fever. Examined in a liquid, the individual bacilli are seen to make vigorous movements. This mobility is dependent upon the thin, filiform fibers at the ends of the comma bacilli discovered by Löffler.

The comma bacilli flourish best at a temperature between 86° and 104° F. (30° and 40° C.). Below 61° F. (16° C.) they cease to grow, but they are not killed even by a greater degree of cold. The free access of oxygen promotes but is not absolutely indispensable to their growth. They multiply very rapidly in liquids—e. g., broth or milk—and they may, under favorable circumstances, retain their vitality for many weeks, while they can be readily destroyed by desiccation. In this again they resemble the genuine spirilla, which can maintain their existence only in fluids. On the human hand the comma bacilli usually die from desiccation after two hours, while on food stuffs in moist storage they keep alive for about eight days, and on moist laundry even fourteen days. The characteristic features of pure cultures can not be described in detail here, but we may observe that the nutrient gelatin

is slowly liquefied by the bacilli. If a few drops of sulphuric acid are added to a fresh bouillon culture, a reddish-violet coloration appears, due to the indol formed from the bacilli and the sulphurous acid.

There can now be no doubt that the sole cause of cholera is infection by the comma bacilli. It has been shown that in every case of genuine Asiatic cholera the comma bacilli are present in the intestine, and that they are never found under any other circumstances. The transmission to animals (guinea pigs) is successful only when the gastric juice has previously been rendered alkaline by means of a soda solution. Injection of cholera cultures into the peritoneal cavity of guinea pigs rapidly effects fatal intoxication from the bacterial proteins. By slow preliminary treatment with small, gradually increased doses of cholera cultures, the animals can ultimately be immunized against the most powerful toxin actions. The blood serum of animals, thus immunized, is agglutinative to the cholera vibrios (see p. 25) and, if injected simultaneously with a fresh cholera culture into a guinea pig's peritoneal cavity, rapidly dissolves the comma bacilli.

Investigation as to the origin of cholera must, therefore, now meet this culminating question: Under what circumstances and through what channel do the comma bacilli penetrate into the human system, and in what manner do they there excite the characteristic processes of the disease? There can be no doubt that among Europeans, and probably everywhere except in India, the cholera is invariably imported. It is equally certain that the dejections of cholera patients, which are rich in comma bacilli, are the chief if not the only agent by which the disease is spread. The bacilli which escape into the outer world with the stools find abundant means to prolong their existence. They continue their growth upon moistened bedclothes, or in water which contains a sufficient amount of organic substances, or in food, such as fruit or milk, or in moist earth; and the ways by which they can in turn enter the system of a healthy human being are infinite in number. It is easy to understand why certain persons —e. g., laundresses and nurses—are more liable to infection than others; and it is equally intelligible that the spread of the disease should often bear a relation to certain outward circumstances. The fact has long been a familiar one, that the cholera almost always progresses along the world's most frequented highways, and that it never travels faster than the means of human intercommunication render possible. This is important, because it shows plainly that the germs of the disease are not disseminated by currents of air. It is easy to understand that the distribution of the disease should sometimes correspond with that of water destined for personal use. The experience of the last few years has again proved most clearly that the drinking water is the chief, although, of

FIG. 18.—(From KOCH.) Comma bacilli from a cholera dejection which had lain for two days on a wet cloth. The S-shaped bacilli are at *a*. 600 diameters.

course, not the only, source for the dissemination of cholera. If an extensive water supply is contaminated by comma bacilli, as was the case in Hamburg in 1892, the disease may suddenly break out with great severity. Sporadic cases usually arise from the use of a contaminated stream which is used for drinking only by a small number of men (sailors, etc.). It is also important to note that flies, which have rested on the evacuations of cholera patients, on soiled linen, or the like, can carry the infectious materials upon food stuffs, etc.

A man falls sick with cholera, of course, not simply from swallowing the cholera bacilli, but because the bacilli remain in the intestine and multiply. It is safe to assume that many men swallow comma bacilli at the time of a cholera epidemic without falling sick at all, or without any but the slightest disturbance, because the comma bacilli are at once destroyed by the acid gastric juice or they develop in the intestine only to a slight extent. Repeatedly, comma bacilli have been found in the solid stools of healthy men, who were in close contact with cases of cholera, and also in the stools of men who had a very mild "cholera diarrhea." These facts are extremely important. They lead to a correct estimate of the infection experiments repeatedly practiced of late on human beings, and, on the other hand, they have a great practical significance in regard to the danger of transmitting cholera.

Most cholera epidemics happen in the summer months. Liability to the disease is very widespread, although some remarkable exceptions are seen. Sex is unimportant. Age has more influence. The disease occurs in sucklings, but, as a rule, is more rare among children than among adults. Elderly people are very apt to take the disease, while of typhoid fever the opposite is true. Most authors lay great stress upon predisposing causes. Among these, taking cold is not so important as are errors in diet and mild attacks of gastro-intestinal catarrh, which are shown by numerous observations to predispose strongly to the disease, because the acidity of the gastric contents is thus diminished, and the retention of comma bacilli in the intestine is thus facilitated. The stage of incubation seldom lasts over one to three days.

Clinical History and Symptoms.—In cholera, as in most acute infectious diseases, the intensity of the illness varies between the extremes of mildness and severity, so that usually a correct interpretation of the mildest cases is rendered possible only by the knowledge that an epidemic exists and by the discovery of comma bacilli. These insignificant cases are called simple choleraic diarrhea. The symptoms are those of a violent acute intestinal catarrh; the dejections are watery, rather large, painless, and number about three to eight in twenty-four hours. There is considerable malaise, complete anorexia, and thirst, and there may already be indications of severer choleraic symptoms —vomiting, slight pains in the calves of the legs, and diminished secretion of urine. Many cases recover after a few days or a week, but in others the first mild diarrhea is succeeded, at the end of one to three days, or rarely later still, by a severe attack of cholera. In such cases we speak of a "premonitory diarrhea of cholera."

The mild form is succeeded in a gradual transition by the cases designated as "cholerine." Cholerine exhibits the symptoms of a violent, rather sudden cholera morbus. It often begins at night. To the diarrhea, which now and then displays even at this time the characteristics of pronounced cholera, vomiting is soon added. The accompanying constitutional symptoms are rather

severe. There is great languor and depression. The voice grows weak, the extremities are cool, the pulse is small and accelerated, painful cramps occur in the calves of the legs, the urine grows scanty and perhaps albuminous. The whole attack lasts about a week or two, before recovery is complete. The course of the disease is not infrequently varied by repeated improvements and relapses.

From these cases of medium severity there is again a continuous line of transition to the pronounced severe form of cholera. Statistics as to the frequency of the separate forms cannot be given, since many of the milder cases escape observation.

The true attack of cholera may begin suddenly with the severest symptoms. As a rule, however, it is preceded, as already stated, by a first stage of brief premonitory diarrhea. This, after one to three days, is replaced with equal suddenness by the severe symptoms of the second or "algid stage," or "cholera asphyxia." Its first symptoms are the abrupt appearance of great bodily weakness, chilliness, and vertigo. The characteristic gastro-intestinal symptoms soon declare themselves.

The diarrhea grows very violent. At short intervals there are copious painless dejections, which at first retain somewhat of a feculent character, but very soon present a characteristic resemblance to rice water, gruel, or whey. A single stool will measure a little less than half a pint (gm. 200). The stools have no color and almost no odor. They are watery, and usually deposit a finely granular, grayish-white sediment upon standing. Their reaction is neutral or alkaline. Only one or two per cent is solid matter, with a little albumen and a relatively large amount of sodium chlorid. In many severe cases the dejections contain more or less blood. The microscope reveals epithelium, triple phosphate, and numerous microörganisms. Of these last, a part are the comma bacilli, and a part are bacteria of putrefaction, etc.

These excessive evacuations are but very rarely absent. They are more apt to fail if death occurs at the end of a few hours—*cholera sicca.*

[In cholera sicca the intestines after death contain the characteristic rice-water material which, perhaps owing to paralysis of the muscular coat, was not expelled during life.]

The appearance of the diarrhea is soon followed by frequent though rarely distressing vomiting. The vomitus consists in part of ingested liquids and in part of an actual transudation through the mucous membrane of the stomach and intestine. Hiccoughs may accompany and follow the emesis.

In addition to these prominent digestive symptoms of vomiting and profuse diarrhea there are complete anorexia and excessive thirst. The tongue has a thick, dry coat. The abdomen is usually flat and soft, or it may be concave and hard. Sometimes we may feel fluctuation in the intestines, due to their being filled with fluid. There is not much real abdominal pain; what there is, is described as a "feeling of heat and pressure" around the umbilicus.

At the same time very severe symptoms develop in other organs. The circulatory system is chiefly affected.

The action of the heart may be excited at the beginning of the attack. The patient complains of palpitation and great precordial anxiety. After a brief time, however, cardiac weakness appears, and continually increases. The action of the heart becomes very weak, and the heart sounds feebler and feebler. The pulse at the wrist grows very small, and is usually somewhat accelerated,

the artery narrow and contracted. In a severe case the pulse vanishes completely after a few hours.

This collapse of circulation makes itself quickly evident in the appearance of the patient. The face and extremities grow cool, and then ice-cold; the complexion becomes partly livid and partly a bluish gray; the lips are almost black. The surface temperature may fall below 95° F. (35° C.), while in the rectum febrile temperatures may often be observed, reaching 102° F. (39° C.) and higher. The eye and cheek grow very hollow, the skin becomes wrinkled, and loses all its elasticity. The voice grows hoarse and feeble (voice of cholera). Respiration is laborious and superficial. The mind may remain unclouded to the end, but usually there is great apathy, and all acuteness of perception is destroyed. Only a few patients are restless and excited. Reflex action is much impaired.

One characteristic symptom is the cramps in the muscles. These are usually very painful, and consist in tonic contractions of the muscles, particularly those of the calf of the leg, but also those of the toes, thighs, arms, and hands. The cramps occur spontaneously or upon the least provocation, last a few minutes, and recur at short intervals. They are to be regarded as a direct effect of poison, and occur, too, in other severe acute enteric diseases, especially cholera morbus.

In a well-developed attack of cholera there is almost invariably oliguria or anuria. The urine, if any be secreted, is concentrated, with abundant sediment, and it very often contains albumen. In many cases not one drop of urine reaches the bladder for days, and this condition persists till death or recovery.

The symptoms thus far depicted, if taken as a whole, represent the algid stage, which seldom lasts more than one or two days. In many cases death occurs during this period. It is ushered in by the tokens of extreme general prostration, and may take place after a few hours, or more frequently in the second half of the first day. But in other cases the "stage of reaction" succeeds. This may be a true compensatory period, leading directly to convalescence. The evacuations become less frequent and more feculent, and the vomiting ceases. The pulse becomes stronger, the cyanosis and coolness of the extremities diminish, and an abundant perspiration is not infrequent. After a few days urine is again excreted. This is almost invariably quite albuminous, and usually contains casts and red blood globules. If convalescence be uninterrupted, however, the urine very soon becomes perfectly normal, and after a week or two recovery is to be regarded as complete.

Departures from this favorable course of the stage of reaction are frequent. Recovery may be interrupted by repeated relapses into the previous condition, sometimes with a fatal result; instead of convalescence, there is developed a severe third stage, usually with fever. This stage ordinarily bears the generic name of cholera typhoid, although it is subject to manifold variations in its clinical symptoms as well as its exciting causes.

Cholera typhoid may present an actually typhoidal general condition with severe fever. There is a considerable elevation of temperature, headache, and dullness. The pulse is full and rapid, the face flushed. The skin, particularly that of the extremities, sometimes presents the so-called choleraic eruption, in the form of an erythema, roseola, urticaria, or the like. This variety of chol-

era typhoid ends after a few days in recovery, or else passes into one of the following conditions:

A second form of cholera typhoid is distinguished by the development of the most diverse local inflammations. Thus, there may be a severe dysenteric or diphtheritic inflammation of the small and large intestine, attended by offensive purulent and bloody stools. Pneumonia is also possible, as well as purulent bronchitis, diphtheritic inflammation of the larynx, pharynx, bladder, and female genitals, parotitis, and sometimes erysipelas and pyæmia. And when we consider that, besides all these conditions, the usual intestinal symptoms, or those of choleraic nephritis, may exist also, it is evident how varied the clinical picture may be. The development of these local affections frequently lays the foundation for numerous sequelæ.

Choleraic nephritis gives rise to the third or uræmic variety of cholera typhoid. The secretion of urine is almost suspended. The region of the kidneys is sometimes sensitive on pressure. The little urine that is still passed contains numerous casts, albumen, and frequently renal epithelium and white and red blood globules. Somewhere toward the end of the first week, or possibly earlier, there are grave nervous symptoms, to be regarded as uræmic: first there is headache and vomiting, then sopor and coma, or delirium and convulsions. Most of these cases are fatal.

Pathology and Pathogenesis.—We are now acquainted with the manifold symptoms and varieties of the disease. If we seek for the pathological changes which control the process, and endeavor to find some correspondence between them and the symptoms, we shall be disappointed. At least, in its early stages, cholera is merely a severe local disease of the intestine. We find the serous layer of the coils of the small intestine rose-red from congestion. The mucous membrane is in a state of catarrhal inflammation: it is swollen, reddened, and at first covered with a layer of tough, transparent mucus; but very soon an abundant transudation flows into the canal, so that the coils of the small intestines are filled with a large amount of clear fluid, looking like rice water or gruel, and so devoid of bile as to indicate the suspension of its secretion. The signs of inflammation of the mucous membrane now grow more pronounced. The solitary follicles and Peyer's patches become swollen, with edges of a vivid red, and frequently there are many small ecchymoses in the mucous membrane. The extensive desquamation of the epithelial lining of the intestine has also been regarded as important, because it was considered as in part the cause of the copious transudation. Still it may be questioned whether the desquamation is not, at least to some extent, a post-mortem change. In yet later stages of the disease the intestinal trouble very frequently assumes a croupous-diphtheritic character. The surface is necrosed and ulcerated in many places, and the contents of the intestine are no longer colorless, but sanious and bloody, with a foul odor.

Otherwise most of the post-mortem lesions correspond to what was obvious at the bedside. The muscles exhibit an early and persistent rigor mortis, and frequently contract in such a way as to throw the corpse into some unusual posture. All the internal organs are remarkably dry, pale, and anæmic. The left ventricle is contracted. The blood lies mostly in the large veins, the right side of the heart, and the cerebral sinuses. It is thickened, is but little clotted, and is said to resemble the juice of bilberries or huckleberries. The spleen is

not enlarged—an exception to the rule in infectious diseases. The kidneys present marked passive congestion, most pronounced in the cortex. The microscope reveals a greater or less degree of parenchymatous nephritis, with great destruction of the epithelium. If death takes place at a rather advanced stage of the disease, the tissues have lost their characteristic dryness, and the most diverse local lesions, including nephritis, may be found to have occasioned death.

If we search for the connection between the pathological changes just described and the cause of the disease, or again between these lesions and the clinical symptoms, the first point to guide us is that the comma bacilli are found only in the lumen of the intestine, and never in the blood or in other parts of the body. The intestinal symptoms are satisfactorily explained by this abnormal state of the intestine, but for all the other grave symptoms we have to seek some special cause. The desiccation which the body undergoes as a result of the excessive liquid dejections cannot fail to affect the tissues, but cannot fully explain the symptoms, for the circulatory disturbances and the cardiac failure at least may develop before large evacuations have occurred. It has also been settled beyond question by means of the newer investigations that precisely the worst symptoms of cholera—especially the cardiac weakness with its resulting algidity as well as the muscular cramps, the subnormal temperature, and probably also the nephritis—are occasioned by the toxic proteins (endotoxins) in the bacteria or by the chemical results of tissue metamorphosis in the comma bacilli. It is an interesting circumstance that the amount and virulence of the toxins formed by the comma bacilli seem to depend on the physical characteristics of the nutrient material on which the culture grows.

As to the complications which occur in the later stages of the disease and which are embraced under the generic name of cholera typhoid, we regard them as mainly secondary. The choleraic process itself does not cause them, but is merely the occasion for their appearance. The examination of the intestines in such cases shows that numerous other varieties of bacteria follow closely upon the comma bacillus, gaining entrance to the system by treading in its footsteps. Cholera nephritis is probably due to various causes. Some forms of nephritis seem to be caused by specific cholera toxins (in analogy with scarlatinal nephritis), while others are probably of a secondary septic nature.

Diagnosis.—A positive diagnosis of cholera can be made only by finding the comma bacilli in the stools. When an epidemic prevails it is often neither necessary nor possible to make the search in each individual case. Sporadic cases or the first cases of a beginning epidemic can and must be diagnosticated only in this way, which gives absolute certainty. For this alone shows the measures necessary for the prevention of the spread of the disease. We cannot give here a detailed account of the bacteriological diagnosis, but we may state briefly that, when cholera exists, the simple microscopical examination of a smear preparation sometimes makes the diagnosis extremely probable.

The examination of the cultures, agglutination, and Pfeiffer's test (*vide supra*) must also be considered. The comma bacilli are also agglutinated by the blood serum of persons who have had cholera.

All those affections whose symptoms are like those of cholera may be confounded with true cholera, especially cholera morbus (*vide infra*); and also

certain cases of poisoning, especially acute arsenical poisoning, may give rise to symptoms wonderfully like cholera.

Prognosis.—The prognosis should always be guarded at the beginning, even if the symptoms be mild, for, as already mentioned, a simple diarrhea may prove to be "premonitory" of a severe attack of cholera. During the real attack the prognosis grows graver in proportion as the case presents the characteristics of asphyxia and cyanosis. The mortality in many epidemics is frightful. All the inhabitants of a house or street may in a brief period be swept away. Accurate statistics are difficult to give. If we count the typical cases alone, the mortality is not infrequently fifty to seventy per cent. In about two thirds of the fatal cases death occurs during the first days of the stage of asphyxia, and in about one third during the second period, known as "cholera typhoid." The influence of the diet and the hygienic surroundings of the patient before his illness is important. A greater proportion of children and old people perish than of the middle-aged.

Prophylaxis and Treatment.—The measures to be taken to prevent the spread of the disease, when it has once started in a place, can be only briefly discussed here. The chief things are the quickest isolation possible of the first cases that occur, the disinfection of the stools and all linen, objects, etc., soiled by the stools, and finally the determination of the source of infection (drinking water, etc.), in order to prevent further infection. The evacuations (stools and vomitus) can be best disinfected with a five-per-cent solution of carbolic acid or with milk of lime, chlorid of lime, etc.; the linen and other objects should be disinfected in a special disinfecting apparatus. It is very important to keep a strict oversight of the drinking water, milk, and all articles of food eaten in an uncooked condition.

In regard to individual prophylaxis, we must remember especially that any slight gastric or intestinal catarrh increases the liability to the disease. Hence, at the time of a cholera epidemic, careful dietetic restrictions are imperatively necessary and every disturbance of the stomach or the intestines, even the slightest, needs the promptest and most careful treatment. It is best wholly to avoid the use of water that has not been boiled, raw vegetables, etc.

It has been proved possible to attain protective inoculation against cholera for one to one and a half years by injecting attenuated cholera bacilli (Haffkine) or by inoculation with small quantities of cholera-agar cultures previously killed by heating or by chloroform vapors (Pfeiffer and Kolle). But to what extent protective inoculation will assume practical importance cannot yet be told.

In the treatment of the cholera attack itself many physicians even now use opium (laudanum or pure opium in powder, gr. ss.–j, gm. 0.03 to 0.05), while others, especially at the beginning of the disease, prefer large doses of calomel (gr. v–x, gm. 0.3 to 0.5, several times), and during the subsequent course they give continued small doses of calomel (gr. ss.–j, gm. 0.03 to 0.05, every two hours). In the algid stage warmth is especially advisable. Hot baths, hot packs, sweat-boxes with dry or moist hot air, embrocations of hot oil, an abundant supply of hot drinks (coffee, tea, mulled wine, broths) are praised by all physicians, and they must certainly be used. The remaining treatment is symptomatic; morphin or ice for the vomiting and embrocations of chloroform oil or subcutaneous injections of morphin for the painful cramps in the

calves. The weaker the heart becomes, the more energetically must we give stimulants (injections of camphor, champagne, caffein, etc.).

Among the newer methods of treatment tried during the last epidemic we may mention subcutaneous infusions of warm (40° C.) 0.6-per-cent salt solution in the infraclavicular region or under the skin of the upper thigh. Half to one liter (quart) of salt solution can be infused several times daily. Intravenous injections have given still better results. Cantani strongly recommends intestinal injections ("enteroclysis") with warm tannin solutions at a temperature of 100° to 104° F. (38° to 40° C.). The fluid contains 5 to 10 parts of tannic acid, 50 parts of gum arabic, and 2 or 3 parts of laudanum to 2,000 parts of water. As an internal remedy lactic acid has been especially recommended (acid. lact., ℥ss.; aq. destil., ℥vij; syr. simplicis, ℥iijss.; 1 or 2 tablespoonfuls every hour or more), also large doses of talcum or bolus alba. All these methods are believed to have shown some favorable results, but none of them has been able to attain general recognition and use.

[The vital importance of the serious treatment of a beginning diarrhea during a cholera epidemic cannot be too strongly insisted on. Rest, simple diet, and a little medication will, in the vast majority of instances, entirely prevent grave consequences. The apparently trifling character of the symptoms is apt to lead people into a false security. Those who can leave an infected district should do so without delay.

With reference to the prevention of an epidemic, a pure water supply and strict cleanliness in its broad sense possess far more virtue than cordons of troops or measures of quarantine. It is more practicable to destroy the soil than to keep out the seed in these days of constant and rapid international communication. The systematic disinfection of all cholera discharges or articles soiled by them should be a matter of course.]

Great caution must be exercised about the diet, not merely during the attack itself, but for a considerable time afterwards. At first we can allow only thin porridge, milk, broths, and toast or rusks. It is advisable to administer dilute hydrochloric acid with the food.

The treatment of cholera typhoid varies greatly, of course, according to the kind of attack. The separate complications should receive their customary treatment.

CHAPTER XIV

MALARIAL DISEASES

(Intermittent Fever. Fever and Ague. Swamp Fever)

Ætiology and Pathological Anatomy.—Malarial poisoning is the best example of what was formerly called a purely "miasmatic" affection. The disease producers are without doubt localized in certain places, in which every human being is liable to become its victim; but if an infected person comes to a place free from malaria and not naturally favorable to its development, there is no danger that he will cause the disease in others. The disease is never caught through contact with the patient. It is not at all contagious; the malarial

poison, after it has once penetrated into the body, has practically no opportunity to escape again in an efficient form from the diseased system into the outer world; but the blood of a patient injected into a healthy person may transfer the disease (Gerhardt and others).

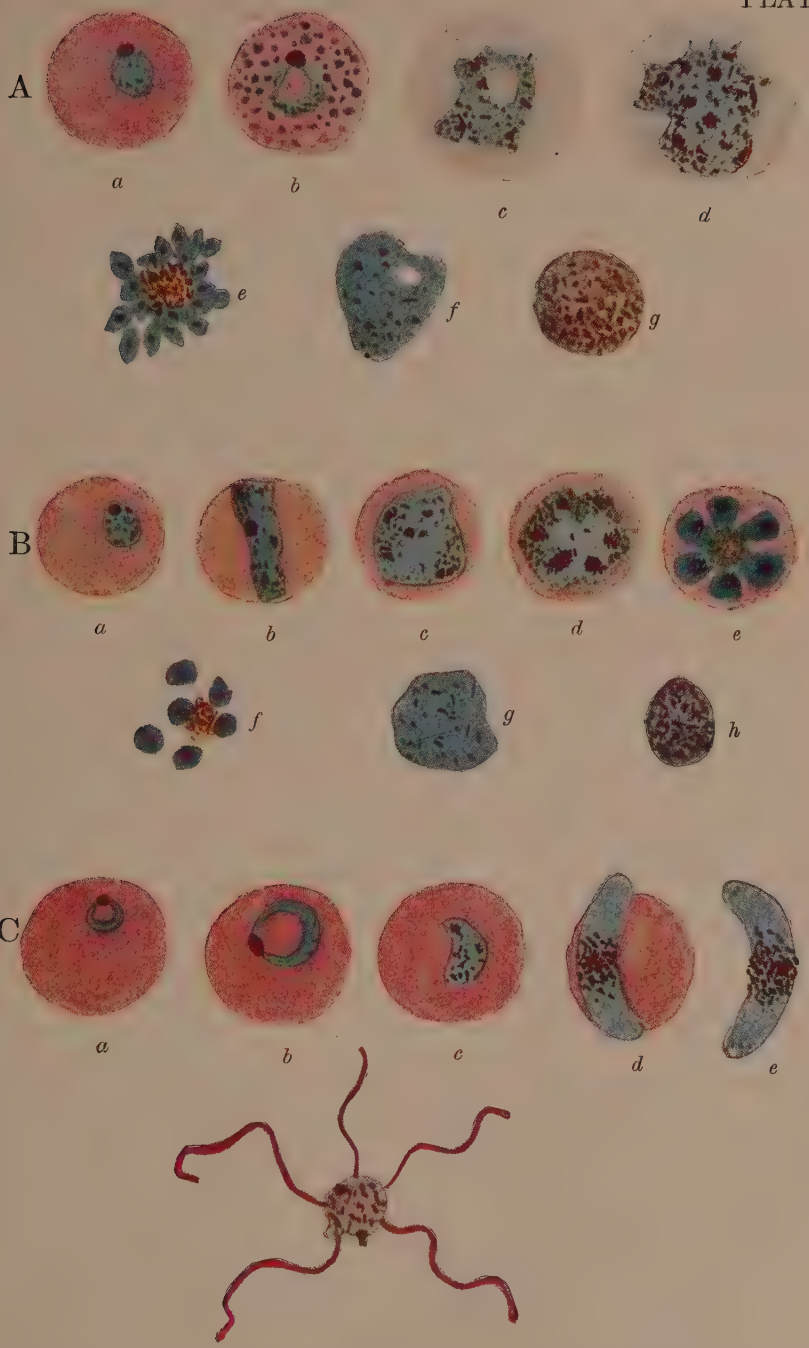
If we except the polar zones, there are few regions where malaria is not endemic in certain parts, at least from time to time, if not constantly. There is, however, great variation in the virulence as well as in the number of cases. Moreover, the disease is in many places becoming less and less frequent because of the general improvement in the sanitary and social conditions. While the common forms of intermittent fever are very frequent in Germany, in numerous places, especially on the shores of the North and Baltic Seas, and also in the alluvial lands of the Vistula, Oder, Elbe, etc., yet the grave forms of the disease are very rare. Other lands are notorious for the severe malarial diseases—e. g., Hungary, the lands lying on the lower Danube, the Roman Campagna, the Pontine marshes, Sicily, and numerous districts in other parts of the world, chiefly tropical.

All these long-known facts, proved by experience, have been explained by the discovery of the specific producer of malaria and the manner in which it is transmitted to man.

The malaria producer was first described by Laveran in 1881, and soon after by Marchiafava and Celli. Since then many other observers, especially in Italy, have investigated the subject and have given us very interesting information about these peculiar parasites. The real process of malarial infection through mosquitoes has, however, only become known to us by Ross's investigations in India, incited by Manson, and by the later work of Grassi in Italy. In London, where malaria never occurs, Manson had healthy persons bitten by anopheles previously nourished with the blood of malarial patients in Rome, and the subjects fell sick of malaria.

All malarial diseases are due to the invasion of the red blood corpuscles by a special kind of protozoa, the so-called malarial plasmodium. The malaria producers are classed among the sporozoa group—i. e., among the so-called hemosporidia, which are present in the blood of numerous animals (frogs, reptiles, birds, mammals). In human malaria, several related but not identical forms must be considered. We differentiate the parasites of the tertian (and quotidian, *vide infra*), those of the quartan and those of the severe, pernicious forms (*vide* table).

The incubation period of the plasmodium of the tertian fever is forty-eight hours. By staining a smear of the patient's blood with methylene blue (and numerous other stains which need not be set forth here), small blue rings can be found in and on the red corpuscles; the rings have a thinner and a crescentic thicker half. In the center of the thinner half there is a small nucleus. These bodies, resembling in shape a signet ring, are called "small tertian rings." After about twenty-four hours the attacked blood corpuscles have become pale, materially larger, irregularly formed. The parasites in them have grown considerably, but still show a somewhat irregular ring form ("large tertian rings"). They contain abundant pigment distributed in very fine granules. Some parasites have wholly lost their ring-like shape and present the most varied amœboid forms. Finally, all parasites grow to larger-sized clumps which occupy almost the entire corpuscle. The pigment in them



A.—Malaria. Large Tertian Parasite. *a–d*, Intracorpuscular developmental forms; *e*, plasmodium undergoing segmentation; *f*, macrogamet; *g*, microgametocyte. (*f* and *g*, sexual types.)

B.—Malaria. Quartan Parasite. (Somewhat schematic.) *a–e*, Intraglobular developmental forms; *f*, free spores; *g*, macrogamet; *h*, microgametocyte. (*g* and *h*, sexual types.)

C.—Malaria. Severe Tropical Form. *a*, small; *b*, large tropic ring; *c* and *d*, intraglobular crescentic forms; *e*, free crescentic form; *f*, flagellated form. (Microgametocyte.)

collects more and more to single clumps and rows, until it finally agglutinates to one or two clumps in the center of the parasites. Now numerous individual stripes appear in the body of the parasite so that it resembles a raspberry. The last capsule of the red corpuscle bursts, and fifteen to twenty-five small spores (gymnospores), created by asexual division, swarm into the blood. The spores penetrate into new red corpuscles, and a new attack of fever ensues.

But besides this asexual spore formation, peculiar processes, preparatory to the other form of propagation—the sexual form—can be seen in some of the full-grown plasmodia of the fresh blood preparation. Some plasmodia develop to the so-called gametæ—i. e., to small male gametæ with hyaline plasma, from which grow out four to eight long, thin, flagella (spermatozoa), and to larger, fine-granuled female gametæ. The fertilization of the female macrogametæ occurs partly in the blood, but still more in the stomach of the female mosquito (anopheles). The further development of the female gametæ, however, only occurs in the body of the mosquito. From the fertilized macrogametæ there form small, crescentically curved worms (oöcinetæ), which perforate into the stomach walls of the insect. After about two or three days they grow up to so-called oöcysts, which after a further two to three days show in their interior numerous small globes (daughter cysts, sporoblasts). Finally, the oöcyst is filled with innumerable small crescentic germs, which in their turn have been generated in the sporoblasts. Now the cysts burst, the crescentic germs get into the peritoneal cavity, and thence by way of the lymphatic stream into the salivary glands of the mosquitoes, where they are demonstrable in enormous numbers about eight to ten days after the insects have sucked the blood. By the sting of such insects crescentic germs are transferred into the blood of the bitten person. From them there develop, in a manner not yet fully known, the malaria plasmodia which cause the febrile attacks.

The parasite of quartan fever shows at first quite the same form as the small tertian rings (*vide supra*). But after twenty-four hours the ring transforms itself into a pigmented band, which goes diagonally across the blood corpuscles and materially expands in the next twenty-four hours. The attacked blood corpuscles otherwise retain their usual size and coloration. Finally, the parasite entirely occupies the blood corpuscle and breaks up. The entire development takes seventy-two hours. With it there occur gametæ formation and sexual propagation in the mosquito's stomach, entirely analogous to what has just been described for the tertian parasite.

The parasite of tropical fever forms small rings which enlarge in the course of the fever ("small" and "large tropic rings"). The blood corpuscles themselves do not become larger and paler. The gametæ of the tropic parasites appear in the form of small moon crescents. We cannot here discuss the numerous interesting details.

By the discovery of the important rôle which the mosquito plays in the spread of malaria, a mass of epidemiologic facts regarding the occurrence of the disease have at once been cleared up. We know that only the anopheles can transmit the disease, not the culex varieties. It is further noteworthy that only the female anopheles subsist on blood, and therefore can transmit the malaria. Only where temperature and moisture conditions satisfy the life needs of the anopheles can malaria develop. Where levees are drained and

the mosquitoes disappear, there malaria ceases. Only where mosquitoes can be the agents of transmission is the importation of the disease by a malaria patient into a malaria-free region possible. As mosquitoes exist only in the warm season, malaria is chiefly a summer disease. It has long been known that in malarial regions it is especially dangerous to spend the night in the open air or to sleep with open windows. This is at once explained by the fact that mosquitoes bite only at night. The mosquitoes do not fly very high above ground; this agrees with the fact long taught by experience that it is particularly dangerous to sleep on the ground, and much less so in elevated places. Everything which protects from the mosquito—nets, fires, etc.—also protects from malaria. When, through a universal, thorough treatment with quinin (*vide infra*), the plasmodia in the blood of the human beings in a district are destroyed, the sickness is more and more restricted, despite the most numerous anopheles.

To all appearances the malarial plasmodium and its various stages of development cannot long maintain a separate existence outside the human body and the mosquito's body. This fact affords the best security that gradually it will be possible to exterminate malaria. The period of incubation, from the time of the infection by the anopheles' bite till the first attack of fever, lasts at least five to six days, but may take as long as twenty-one days. Susceptibility to the disease is very general. No race, no age, no sex is immune from malaria.

The investigations with regard to the plasmodium of malaria have also explained the long-recognized fact that in chronic malaria large amounts of pigment collect in the internal viscera. The pigment is a metabolic product of the parasites and arises from the hemoglobin of the attacked red blood corpuscles. The liberated pigment is taken up by the leucocytes and deposited in the inner organs. These deposits are most abundant in the spleen, which in the chronic varieties of the disease develops into a firm, hard tumor; but they are also found in the bone marrow, liver, brain, and kidneys, leading finally in the liver and the kidneys, in frequent instances, to processes of chronic degeneration and inflammation. It is especially noteworthy that in those patients who present the most marked cerebral disturbances (pernicious comatose fever, *vide infra*), the cerebral capillaries are found to be completely occluded with pigmented plasmodia.

[Periodical fever is very widely distributed in the United States, and in the southern portions occurs in severe, though not in the severest, forms. Some regions which were formerly free from it are no longer so, and, *vice versa*, some regions which were greatly subject to it are now exempt.

The hopes which have been entertained in some quarters that malarial regions might be rendered healthy by large plantations of the Australian eucalyptus globulus, a rapidly growing tree which absorbs immense quantities of water, do not seem likely to be realized in the light of French experience in Africa, and in that of the Trappist monks in Italy.

The following details may be of use to those who are not familiar with the method of blood examination: The finger tips should first be scrubbed with a nailbrush, and soap and water, and then washed in alcohol or ether, to obviate the danger of mistaking minute particles of dirt for the pigment-granules derived from the destruction of red blood corpuscles. A portion of

a drop of blood is then to be received on a scrupulously clean object- or cover-glass, and squeezed out between the two, so that a thin layer of blood with separation of the individual corpuscles is secured. The amœboid forms of the plasmodium retain their movements for a number of minutes in a warm room, much longer, of course, with a warm stage. The presence of pigment-granules in the red corpuscles may be the readiest indication of the presence of the organism. The crescentic forms are found more especially in chronic cases which have been under treatment by quinin, as is stated by Osler. Many of the other forms can be found in the blood only during a paroxysm.

In dried and stained specimens it is also possible to detect the organisms, but amœboid movement is, of course, lost.]

We shall consider below chiefly the common forms of intermittent, such as appear in Germany, contenting ourselves with a very brief description of the severer forms.

Varieties of Malarial Disease.—1. Intermittent Fever.—This is the simplest and most frequent form, and has for its especial characteristic the relative brevity of the febrile attacks, which almost always exhibit a remarkably uniform type. A febrile attack of this kind is frequently the very first symptom of the disease. In other cases the paroxysm of fever is preceded by a prodromal stage lasting several days, during which the patient feels languid, has no real appetite, complains of headache and pain in the back of the neck and in the limbs, and often even thus early presents a slightly yellowish complexion and an enlarged spleen.

In the typical attack of intermittent fever there are three stages. The attack begins with a chill. There is pronounced malaise, attended by intense chilliness and more or less shivering. The skin is cool and pale, the face may be somewhat livid. The temperature of the interior of the body is elevated, and rapidly rises higher. In by far the greater number of cases the attack occurs in the morning, or at least before noon, and but seldom later in the day. This cold stage varies greatly in length, usually lasting an hour or two.

The chilliness is followed by the hot stage. The skin grows burning hot, the face flushes, the pulse, which was before small, becomes full, and the action of the heart is excited. At first the temperature continues to increase, and reaches in this stage its maximum for the attack. It is exceptional for it to remain under 104° F. (40° C.), and by no means rare for it to touch 106°, or even 107° F. (41° to 41.5° C.). This stage almost always lasts longer than the preceding, generally about three to five hours. The temperature may begin to fall as early as the latter part of the hot stage, but may persist till the beginning of the third stage.

In this sweating stage the skin grows moist, and there is soon a profuse general perspiration. The patient begins to feel much better. In a few hours the temperature usually becomes normal, and, after lasting in all about eight to twelve hours, the attack is over. It may be shorter or rarely longer. Usually, however, the temperature keeps on sinking slowly, so as to be still subnormal even on the next morning, perhaps not above 97° F. (36° C.).

There are certain peculiarities in the temperature curve which we have ourselves observed. The elevation of temperature is almost invariably more rapid than its decline. The rise is most abrupt during the first hours of the

cold stage, and slower during the first portion of the hot stage. The ascent is but very seldom interrupted. During the hot stage, when the fever is highest, in the neighborhood of 106° F. (41° C.), there are not infrequently two little summits to the fever curve, if the temperature be taken at short intervals. But the temperature may for hours remain the same. The temperature generally begins to fall some little time before the perspiration is evident. The decline is slow. It may be perfectly continuous, or it may be interrupted by fresh elevations, which are sometimes slight and sometimes considerable. In many cases the descent is by steps, the temperature remaining the same for half an hour or an hour, and then abruptly falling a couple of degrees and remaining for a time at this new level.

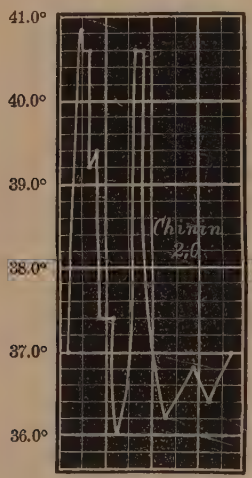


Fig. 19.—Quotidian intermittent fever.

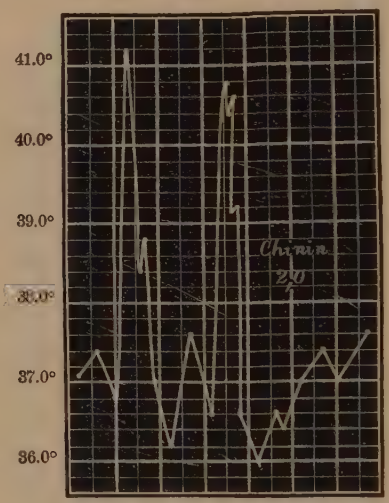


Fig. 20.—Tertian intermittent fever.

The chief characteristic is not, however, the nature of the single attacks, but the peculiar manner of their repetition. If the case is not under treatment, the single attacks keep recurring for a long time every second day. This type of tertian intermittent fever (*cf.* Figs. 19 and 20) is probably the most frequent. Not infrequently daily attacks of fever occur (quotidian intermittent). But this quotidian fever is nothing more than a double infection with tertian parasites, the attacks being caused by two different generations. If there are two attacks in one day, a rare event among us, we have a double quotidian. If there is a violent attack every second day, and on the intervening days there are milder attacks, it is a case of double tertian. Very often the attacks do not recur at just the same time of day, but a few hours earlier each time. Less frequently they are later. This peculiarity is expressed by the term "anticipating" or "retarding," as the case may be—e. g., a retarding tertian ague. In cases of long standing, the paroxysms may finally lose all regularity, so that the fever is described as "erratic."

All these irregularities of the fever are certainly related to variations in the development of the parasites and their several generations. The typical intermittent quartan fever, in which the attacks appear only after seventy-

two hours each time, owe their occurrence to a special variety of the parasites (the quartan parasites, *vide supra*).

Next to the febrile attacks, the swelling of the spleen is the most constant and important symptom. It is usually considerable and capable of demonstration by percussion and palpation. At first the tumor increases with every fresh attack, and diminishes but little during the intervals. After the patient is freed from his attacks of fever the spleen may continue enlarged for some time. It is tender on pressure. [Certainly in many cases tenderness is absent.—V.] The liver may likewise be swollen, but this is less constant and also less important.

Certain changes in the skin are very characteristic, chief among which is a peculiar yellowish-brown discoloration. This is due to an abnormal deposition of pigment in the skin. Herpes on the lips or nose is seen very frequently during the attacks. We have seen one case of herpes on the cornea. Mention has also been made of urticaria, purpura, and other eruptions.

Other internal organs than those already spoken of are rarely much disturbed. One symptom should be mentioned, which we have ourselves seen several times, viz., a quite marked acute dilatation of the heart during the attack. There were no bad results, and the normal condition was soon reëstablished. We may hear during the attack functional cardiac murmurs of a blowing character. Thoracic examination, particularly if made during the attack, may afford signs of a dry bronchitis. Sometimes there is considerable diarrhea, or other evidence of intestinal derangement. Catarrhal jaundice is confined to the severer cases. Sometimes the urine has a moderate amount of albumen. Genuine nephritis is met with only in the graver varieties of the disease. The increased excretion of urea on the days of the fever results, as in any fever, from the increased destruction of albumen. Severe pain in the cervical and upper dorsal vertebræ is regarded as characteristic of intermittent.

Besides the typical attacks, rudimentary and modified ones are not rare, in which the separate stages are ill defined, or in part wanting. We are most apt to see this in cases which have been already treated with quinin. Children do not have a true rigor. They merely become pale or livid. They may present marked nervous symptoms.

2. Pernicious Intermittent Fever.—The malarial fever caused by tropic parasites, briefly described above, also runs its course in single attacks, but these often last for a long time and occasionally occur in such rapid succession that an almost continual fever results. The single stages of the febrile attack are not so sharply defined as in the ordinary intermittent form. The splenic swelling develops only in the later course of the disease, while from the beginning a severe anæmia is prominent. The severer forms of pernicious fever appear especially through relapses or with renewed infections. Pronounced nervous symptoms are most frequent. There may be unconsciousness, coma, delirium, or epileptiform or tetanic convulsions. None of these symptoms persists longer, as a rule, than does the common sort of an attack, and in a favorable case they vanish completely when the sweating, which is usually profuse, begins. The great danger comes from the recurrence of the attacks. A second form of pernicious intermittent fever causes violent gastro-intestinal symptoms, which may almost exactly imitate the algid stage of cholera, with vomiting, diarrhea, and collapse; or there may be severe cardialgia, dysentery,

and the like. In the so-called pernicious intermittent with jaundice, intense jaundice appears during the attack, with vomiting and diarrhea, and sometimes the gravest nervous symptoms. Related thereto is the pernicious biliary hemoglobinuric fever, a severe form of malaria especially prevalent in West and East Africa, characterized by icterus, severe gastric symptoms, and marked hemoglobinuria; hence the name "black-water fever." As Koch has shown, similar phenomena can be caused in predisposed persons by large doses of quinin.

[The pernicious form occurs in isolated cases wherever the ordinary variety of the disease prevails, but is much more common in the Southern and Western States, and there varies in frequency in different years. Periodicity in the attack is not always observed. The pernicious character is not always manifested in the first attack, one or more mild paroxysms being often precedent. In this country the algid form of pernicious periodic fever is often called "congestive chills," and this form is more common than the comatose or another form mentioned by the author—the hemorrhagic. In the latter the blood escapes from the kidneys, and less constantly from the mucous membrane. During the civil war the mortality among the white soldiers of the United States army from pernicious malarial fever was 23.91 per cent.]

3. Chronic Malarial Cachexia.—In the true malarial regions individuals who have frequently suffered from pronounced intermittent and remittent malarial fever may also acquire a chronic diseased condition, appearing sometimes under most diverse forms and being due to the chronic malarial infection. These patients usually show marked anæmia and a decided yellowish malarial complexion. Occasionally real febrile attacks do not occur at all, but there exist only symptoms of general debility, anorexia, nausea, tendency to diarrhea, or, more rarely, constipation, a feeling of fullness in the head, insomnia, frequent perspiration, muscle and joint pains, dyspnœa, palpitation, etc. In other cases these symptoms are aggravated. Severe nervous phenomena, as trembling, paralyses, psychic disturbances, intestinal symptoms, or icterus may occur. Dropsical conditions develop, and epistaxis, ecchymoses, and scorbutic symptoms have been observed. Examination of the inner organs often shows large, firm, splenic tumor and hepatic enlargement. Ascites occurs but rarely. In some cases irregular febrile conditions still occur (chronic malarial fever), but in their blood plasmodia are demonstrable only with difficulty and in small numbers. Finally, secondary diseases are possible—e. g., tuberculosis, amyloid, or dysentery—and these may prove the immediate cause of death. The milder forms may be cured, but seldom unless the patient abandons forever the malarial district.

4. Masked Intermittent.—This is the designation of cases in which, although there is no fever, certain other disturbances arise in regular intermittent attacks. Chief among these is neuralgia. Its favorite seat is the supra-orbital branch of the trigeminus. It may occur in the other branches of the same nerve, or in the sciatic, the anterior crural, the nerves of the brachial plexus, and elsewhere. Cardialgia and enteralgia may occur in the same way. These attacks last from thirty minutes to several hours, and are frequently associated with all sorts of constitutional symptoms, but, as we have said, are afebrile. There may be a splenic tumor, which aids diagnosis; but this sign may be wanting.

Numerous other intermittent disorders besides neuralgia have been described as masked intermittent. The list includes gastric and intestinal disturbances, cramps, bleeding, etc. We must add, however, that those who have described diseases of this sort, some of which seem strange enough, have not always been as critical as they ought, and that the connection between many cases of "masked intermittent" and true malaria is more than doubtful, particularly as the demonstration of the plasmodium in the blood in cases of so-called "masked malaria" is almost never possible.

[5. Typho-Malarial Fever.—This is a term which was formerly in use, but it is not a distinct disease. It expresses a combination in the same individual at the same time of the effects of the special poison causative of each affection. Typhoid being a continued fever, its complication with malaria results in a pyrexia of a remittent type. The combination of the two occurs in malarial regions, especially in the Southern States, and may be seen in nonmalarial regions in the persons of those in whom malaria, contracted elsewhere, is in a more or less active state.

The characteristic symptoms of the two diseases are intermingled, those of typhoid, the graver disease, usually predominating. The history of the case and careful observation of the symptoms will generally clear up any doubts felt as to the diagnosis in the early stages. It would naturally be supposed that the combined affections must produce an illness more severe in character and more unfavorable as regards prognosis than belongs to simple typhoid. Such does not, however, seem to be the case. Woodward's statistics show that the mortality of uncomplicated typhoid was far greater among the white and colored troops alike during our late Civil War than was the mortality of typho-malarial fever.

The treatment as regards the typhoid fever differs in no way from that suitable for cases of the ordinary affection; the periodic element demands the management appropriate to simple intermittent or remittent fever.]

Diagnosis.—It is often very difficult to diagnosticate a case of intermittent fever at the first visit, particularly in a place where malarial poisoning is infrequent. The history of the case is not always enough to put one on the right track; and a single examination of the patient may prove equally negative in its practical results, whether it is made during the febrile stage or in the interval. Continued observation, however, will almost always disclose the regularity of the febrile attacks, the splenic tumor, the characteristic complexion, and the herpes; and our diagnosis becomes evident. Still, it is not very exceptional for an intermitting fever to be taken at first for an intermittent malarial one, while eventually some quite different disease is found to produce the symptom. Pyæmia may give rise to mistakes of this kind; also purulent phlebitis, acute ulcerative endocarditis, and even tuberculosis. We should be very cautious in making a hasty diagnosis of "irregular intermittent fever." Our own experience has taught us that almost invariably the case turns out to be something else. Where there is doubt we may, in addition to a careful consideration of all the symptoms and a thorough physical examination, be aided by the therapeutic action of quinin (*vide infra*). If a high fever of intermitting type is affected by large doses of quinin but temporarily if at all, then a diagnosis of malarial intermittent fever is rendered

doubtful. An absolutely certain diagnosis can be made only by finding the plasmodia in the blood under the microscope.

After due practice and by the use of lenses of sufficient magnifying power, the parasites may frequently be recognized in the unstained blood. The preparation should be thin. Staining with methylene blue and eosin brings out the structure more clearly. We have already said that the greatest skepticism is requisite in the diagnosis of "masked intermittent."

Treatment.—Malarial poisoning is one of the few diseases upon which we can make a direct attack with assured success. In quinin we possess a remedy which probably acts upon the very cause of the disease, and its therapeutic efficacy is undisputed. Quinin is therefore the sovereign remedy in all forms of malarial poisoning, and is often the only drug employed. In the mild cases, which are the only kind that occur in Germany, we do not usually give the remedy upon the instant that the patient comes under treatment. It is best to wait for one or two attacks, partly to make sure of our diagnosis, and partly to learn what the type of the fever is, whether quotidian, tertian, anticipating, or recurring at the same hour; and in most cases this delay works no harm to the patient. During the attack itself there is seldom any use in special treatment. Of course, the patient must stay in bed and be kept warm during the cold stage, and have lighter coverings during the hot stage. During the afebrile interval the patient may be up if he feels strong enough and is careful. Quinin (hydrochlorate or sulphate) is given about five or six hours before the next attack is due. It is best to administer one large dose of 20 to 30 gr. (gm. 1.5 to 2), either in solution or in capsules of 7 gr. (gm. 0.5) each. If the quinin be given in powder or capsules, it is a good way to follow it with a few drops of muriatic acid, to promote its solution in the stomach. Often one large dose prevents the next attack. In other cases it does occur, but with less subjective disturbance, no chill, and more moderate fever. We must then give another large dose before the second attack is expected. If the attack does not take place, then we may give for several days quinin to the amount of 8 gr. (gm. 0.5) *per diem*. After all, relapses are possible, even at the end of several weeks; but they yield readily to quinin.

The substitutes for quinin proposed in various quarters—conchinin, cinchonidin, euquinin, etc.—have proved to be less effective.

In the treatment of pernicious intermittent fever, of the masked forms, of the remittent and continued fevers, the chief remedy is likewise quinin, given in sufficient doses. Baccelli has shown that sometimes in pernicious fever the direct injection of quinin into a vein may save life. Much less reliable is methylene blue, four to six times daily, gm. 0.1 (gr. jss.), in gelatin capsules. In all cases of considerable duration it is also of the greatest importance to remove the patient from the malarial region, if it can possibly be done. This often proves to be the only way to avoid relapses and attain a perfect cure.

In cases of longer standing, quinin sometimes loses its power. Then we resort to arsenic. It is frequently employed in malarial cachexia and in intermittent neuralgia, either alone or combined with iron. We give gtt. v to viij of Fowler's solution two or three times a day in water. It is still better to give pills of arsenious acid containing $\frac{1}{30}$ to $\frac{1}{20}$ gr. (gm. 0.002 to 0.003) and gradually increasing to a daily dose of $\frac{1}{6}$ to $\frac{1}{5}$ gr. (gm. 0.01

to 0.012). That careful symptomatic and dietetic treatment is also essential is a matter of course.

[There is nothing to be gained by allowing a patient to have an unnecessary chill. If there is a reasonable probability that his paroxysms are due to malaria, a prompt effort should be made to cut them short. Four hours is the shortest time that it is safe to allow for quinin by the stomach with probability that the expected chill will be prevented. The drug acts much more promptly when given hypodermically. The hydrobromate is preferred by some to the sulphate for subcutaneous use on account of the necessity of using acid to dissolve the latter, and the consequent risk of abscess. Such a risk should have no weight if the physician has any suspicion that he has to deal with the pernicious form of the disease. If the stomach is irritable, the remedy can be given by enema. Quinin can also be given by suppository, though it may thus produce some irritability of the rectum; but the impossibility of disguising the bitter taste of the remedy or of making children swallow capsules renders this a valuable means of treatment sometimes in infants and young children.

Warburg's tincture is an antiperiodic which does good service in cases which do not yield to the ordinary methods of treatment.

The hypodermic injection of pilocarpin is reported to abort an impending chill.

Some prefer divided and smaller to the single and large dose of quinin or one of its substitutes, a difference of view which is of minor importance.

In the remittent forms boldness in the use of quinin is required. Cinchonism should be induced as promptly as may be, and maintained to a mild degree for several days; the quantity of the drug can then be gradually diminished.

The treatment of the pernicious forms of periodic fever presents itself under three main heads:

1. The prevention of pernicious paroxysms.
2. The treatment of the paroxysms when present.
3. The prevention of a recurrence.

1. We have seen that very frequently the pernicious character is manifested after the occurrence of one or more mild attacks; consequently, in localities and seasons marked by the occurrence of grave cases it is an imperative duty to treat every mild case promptly and energetically, a course which unquestionably saves many a life.

2. The management of the paroxysm differs according to the form which it assumes; in other words, is largely symptomatic. Bemiss (Pepper's "System of Medicine") says: "The cure of a congestive chill is one of the most difficult problems the physician can possibly encounter." Heat externally, opium and chloroform by the mouth, and morphin and atropin subcutaneously, nutrition by the stomach or rectum, according to circumstances, and alcoholics if the action of the heart is feeble, are the measures of widest application.

Whatever the type of the attack, a weak heart calls for alcoholic stimulation. Cinchonism is always to be induced as rapidly as possible.

In the comatose form it is to be remembered that the cerebral and other nervous symptoms are not due to congestion, but probably to a combination of the malarial and secondary blood poisons. To quote Bemiss again: "Efforts to nourish the patient must never be relaxed. One must see many of these

cases before he can realize how often they recover from conditions apparently hopeless when promptly treated and properly nourished."

The hemorrhagic form, like the others, demands cinchonism and careful nutrition, but also hemostatics. Purgative doses of calomel are indicated in some cases of each form, but should not be given in a routine manner.

3. Prompt cinchonism is the chief means of attaining the third aim of treatment. Removal to a healthy locality should be secured if possible, and the general condition of the patient requires careful attention.

It remains to add that those going to a malarial region can often avoid contracting the disease by taking advice of a local physician as to hygienic precautions, and by moderate divided doses of quinin.]

CHAPTER XV

DENGUE

[This affection has never appeared in Germany, and hence, doubtless, was omitted by the author. The name "dengue" is supposed to be a Spanish corruption of dandy, the term dandy fever having been applied to the disease by the West India negroes on account of the stiff carriage of those affected by it. Another name is "break-bone fever."

The disease generally appears in epidemics, and is almost exclusively confined to tropical and semitropical countries. In 1780 an epidemic supposed to be dengue prevailed in Philadelphia, and outbreaks have occurred repeatedly in the Southern States in the last hundred years. In 1880 Charleston, Savannah, New Orleans, and other Southern cities were visited by it. It is prevalent in Texas, and has recently occurred in the Philippines.

Ætiology.—As to the causation but little is known. Those who have had opportunities of studying the disease consider it both contagious and infectious, and the inference that it depends on a specific germ is readily suggested. It seems to prefer low lands along the seashore, and to be influenced by meteorological conditions in that it generally prevails during the summer and disappears as cold weather comes on. Neither age, sex, nor condition affords any protection from the disease; it was thought by Dickson that one attack generally confers immunity for life.

Pathology.—The disease is so rarely fatal that few opportunities have been afforded for its post-mortem study. So far as is known, it has no peculiar lesions. The prominence and the character of the muscle and joint pains have led some observers to think the affection related in some way to rheumatism.

Symptoms and Course.—The onset is usually sudden, but a pronounced chill is said never to occur. Prodromata similar to those of other infectious diseases are sometimes observed, but the first symptom is very often pain and stiffness in the muscles and joints, with frequent swelling of the latter. The large and small joints are equally affected, and the pain is increased by motion. With the pain there is a rise in temperature and in the frequency of the pulse. The pain is apt to increase during the first two or three days and disappear

on the fifth, but irregular remissions are liable to occur. As the thermometer falls the pain and other symptoms diminish, but reappear in full force with any subsequent rise. During the later days of the disease a skin eruption appears on the upper part of the body, and in severe cases becomes general in about two days. This eruption is very variable in character; it may appear simply as an erythema, or simulate the eruptions of scarlet fever, rubeola, lichen, or urticaria; it is apt to be associated with well-marked heat and itching of the skin. In mild cases the eruption is evanescent or absent. Swelling of the lymphatic glands is not uncommon; in severe cases the mucous membrane of the mouth and nose is reddened, and hemorrhage from the outlets of the body has been observed. Pregnant women are apt to miscarry. Delirium is rare in adults, but common in children; the face is generally flushed, and the eyes are injected; the tongue becomes increasingly coated as the disease progresses, the appetite is lost, nausea is not uncommon, vomiting is rare. The bowels and the kidneys present no constant or distinctive symptoms.

In mild cases recovery is sometimes rapid; sometimes, and especially after severe cases, convalescence is very tedious, the muscular and articular pain and stiffness passing off gradually, and the glandular swelling lasting for weeks. Copious skin eruptions are followed by desquamation.

Diagnosis and Prognosis.—As to diagnosis there can seldom be any difficulty during the prevalence of an epidemic. The first cases are the only ones which are liable to be mistaken, and even their nature cannot remain long in doubt. The prognosis is uniformly good.

Treatment.—We are acquainted with no agent capable of aborting or cutting short the disease; nor is there any known measure of prophylaxis except for an individual to keep away from those places in which the affection is known to prevail.

The treatment of the attack is simply symptomatic; notable pain calls for opium in some form. Quinin has not seemed to be of service. Debility following the attack demands suitable alimentation and tonics.]

CHAPTER XVI

YELLOW FEVER

[This disease is not a visitant of Germany, but its consideration cannot be omitted from a text-book on the practice of medicine for use in America. In the following description the aim will be to bring out the more important features of the disease, while for fuller details the reader is referred to larger works and monographs.

Ætiology.—Yellow fever is an acute infectious disease, confined within certain geographical limits, and occurring chiefly in epidemics of greater or less extent. In certain localities, notably Havana, Rio de Janeiro, Vera Cruz, and other Spanish-American ports, it has until recently been endemic. The influence of temperature is well established; the disease prevails, namely, during the summer or the warm season, and is abruptly arrested by one or

two decided frosts; dampness is favorable to it. That it depends ultimately on a special cause and does not originate *de novo* are undisputed facts. The specific organism seems to be of ultramicroscopic minuteness, but separable, however, from the blood-serum by filtration. Infection appears to be more active at night than during the day, prefers low-lying districts, and in them hugs the ground to a certain extent. It is limited to the sea coast and to altitudes of less than 1,000 feet. Bad sanitary conditions are most important accessory causes of the disease. It is a remarkable fact that in large cities the infection may be of great virulence, but confined to a limited district or districts, by carefully shunning which unprotected persons are comparatively safe. An infected area is apt to extend itself, but the progress is slow, and is interrupted by streams of water, high walls, or simply streets.

In 1881, Finlay, of Havana, suggested that yellow fever might be transmitted by mosquitoes. In 1900 the Yellow Fever Commission of the United States Army, composed of Drs. Reed, Carroll, Lazear, and Agramonte, demonstrated that a certain mosquito, *Stegomyia fasciata,* conveys the disease from one human being to another. An interval of at least twelve days seems to be necessary for the infected mosquito to become virulent. It was also demonstrated that articles of clothing and bed linen soiled by the sick have no power to spread the disease. These facts have been amply confirmed by the success obtained in Havana and Panama in suppressing yellow fever by exterminating the mosquito.

As a rule, one attack of the disease renders the system of that person insusceptible forever after; the natives of a yellow-fever district are far less liable to contract the disease than are those who move into the district from elsewhere, probably because they have had the disease in a mild form in childhood. The negro race is susceptible to the disease, though in a less degree than the whites, and in the colored the affection is far less fatal. Neither age nor sex has any special bearing on susceptibility. The stage of incubation is usually three or four days. Inoculation experiments showed an incubation period varying from forty-one hours to five days seventeen hours.

Pathological Anatomy.—The disease involves no constant and peculiar lesions. In rapidly fatal cases, congestion and often hemorrhage are found, especially in the nervous system, liver, kidneys, and digestive tract. In fatal cases of longer duration more or less parenchymatous degeneration is found. A fatty degeneration of the liver is quite common, and imparts a yellow coloration to the organ, giving rise to the terms *café au lait,* or boxwood liver. Jaundice of the skin and tissues generally is also observable after death, and depends upon causes in no way connected with mechanical obstruction to the flow of bile into the intestine during life. Splenic enlargement is conspicuous by its absence.

Course and Symptoms.—The onset of the disease is generally sudden, but it may be preceded for a few days by malaise and other signs of general constitutional disturbance; the initial chill is seldom severe, reaction following soon and the thermometer rising to 102° to 105° F.; hyperpyrexia is rare. The pulse rate does not increase proportionately with the fever. The face becomes flushed and the eyes injected and watery; a slight jaundice at this early stage is very characteristic; headache and pain in the back are early

and usually very prominent symptoms. The bowels are confined; the tongue is apt to be clean if it was so before the disease came on; the stomach is usually somewhat irritable, and there may be vomiting; moderate epigastric tenderness is common; the mind remains clear, as a rule, but delirium is not very uncommon, and in children a convulsion may usher in the attack as in other acute infectious diseases; slight albuminuria is apt to occur as early as the second, third, or fourth day. This hot or febrile stage may last from twelve hours to several days. The pulse generally declines in frequency before the disease has reached its maximum. As the fever disappears the other symptoms vanish also, and the second, or "stage of calm," begins. From this point recovery may be rapid and uninterrupted, the whole disease consisting of but a single febrile paroxysm of greater or less intensity and of short duration.

In grave cases, and gravity is often foreshadowed in the first stage by marked capillary congestion of the surface of the body irrespective of the intensity of the other symptoms, and after a stage of calm lasting for some hours, but rarely exceeding twenty-four, more distinctive symptoms appear. The pulse is very compressible, the surface of the body is cool, vomiting occurs or becomes more prominent, and hemorrhage is now apt to take place. The escape of blood into the stomach, its retention and the changes which it there undergoes, and its subsequent expulsion, explain the dreaded and characteristic symptom known as "black vomit." Tarry stools sometimes are observed. Hemorrhage elsewhere is also common, occurring from the gums, the nose, eyes, uterus, kidneys, into the skin, etc. Albuminuria with casts is very common. Jaundice, sometimes of a lemon-yellow hue, comes on, and is rarely lacking in severe cases. From this symptom the name of the disease is derived.

In cases marked by more or less complete suppression of urine, coma and convulsions, probably largely uræmic, come on. Some severe cases are of the "walking" type, the patients going about while the malady is far advanced, or even up to the time of death. As a rule, however, muscular prostration is marked.

If the disease does not prove fatal in this third stage, convalescence comes on more or less gradually, and is followed by complete recovery; relapses, however, occasionally occur.

The duration of the affection is variable, but, on the whole, short, usually being less than a week.

Diagnosis.—In mild cases the symptoms are not distinctive, and the diagnosis at the commencement of an epidemic is not likely to be reached except by an experienced observer, and even by him more or less conjecturally. During an epidemic the severe lumbar pain, the headache, the suffusion of the eyes, the early jaundice, the disproportionately slow pulse, the early albuminuria, and the moderate gastric irritability are all-sufficient for diagnostic purposes. Severer cases are also marked by capillary congestion of the surface of the body, and the third stage with the black vomit, hemorrhage, jaundice, slow pulse, scanty urine, and prostration is characteristic. Of course, all the above symptoms are not present in every case. One disease which might give rise to confusion is remittent fever with jaundice. This affection has a different temperature curve, is not confined to the yellow-fever zone, has specific

organisms in the blood, is controlled by quinin, and is not accompanied by black vomit.

Dengue and yellow fever have points of resemblance. In yellow fever, however, the pulse is slow in proportion to the temperature. In both diseases there are severe pains, but in yellow fever these are muscular, while in dengue they seem to involve the bones and joints, and the joints are stiff and sometimes swollen. Jaundice is much more common in dengue than in yellow fever, and it appears earlier. An eruption is very unusual in yellow fever and rather common in dengue. Vomiting and nephritis are the rule in yellow fever and exceptional in dengue. Hemorrhages are much more frequent and severe in yellow fever than in dengue.

Prognosis.—This varies in any given locality with the character of the prevalent epidemic. The death rate is sometimes very high, sometimes moderate; it is greater in hospital than in private practice.

In the first stage of the disease the chief element in the formation of the prognosis seems to be the presence of marked and general capillary congestion of the skin, a symptom which foretells a severe attack. The absence of this symptom is rather less important than its presence. Cases may turn out to be severe in which it is lacking. The frequent deceptiveness of the stage of calm is to be remembered.

Yellowness, black vomit, and suppression of urine are symptoms denoting the greatest gravity, but do not justify the complete abandonment of hope.

Treatment.—There are no means in our power of aborting the disease. Prevention is to be attained by banishing the mosquito both from the sick and from the whole region.

The earlier proper treatment can be instituted the better. Absolute rest and good ventilation are the first requisites. The patient should not be chilled. Emetics and cathartics are not indicated by the disease itself; the condition of the stomach and bowels should be inquired into, and indigestible food or an accumulation of feces should be removed if present. A hot mustard foot bath early in the attack is useful. For the lumbar pains, phenacetin or morphin is indicated. Sinapisms, or other similar external counterirritants, with ice internally, and hydrocyanic acid, cocain, or chloroform are serviceable against gastric irritability. High fever is to be combated by cold spongings, the wet pack, and the cold bath. For hemorrhage, styptic remedies may be used, though it is doubtful whether, when given internally, they are really of much benefit. Of course, no medication is to be resorted to which is likely to heighten a tendency to emesis.

Sternberg's formula for internal use is said to be valuable for quieting the stomach and promoting diuresis. It is as follows: Sod. bicarb., gr. cl; hydrarg. chlorid. corros., gr. ½; aquæ puræ, ℥ ij. M. S. Three tablespoonfuls, ice-cold, hourly.

Suppression of urine is to be met by dry cups to the loins, diuretic remedies internally if the condition of the stomach allows, or the hot-air bath. The results of pilocarpin are disappointing according to Bemiss, who states that he has seen good effects from large enemata of water, preferably cold, if there be notable fever in these cases. Prostration is an indication for the use of alcoholic stimulants, among which iced dry champagne ranks high. It will be

seen that the treatment is entirely symptomatic. The disease is self-limited, has a short course, and the patient will recover if he can be kept alive until the poison is exhausted. During the attack and until convalescence is thoroughly established the management of the diet is all-important. Small quantities of the most easily assimilable food may be given at short intervals if they are tolerated by the stomach; if not, alimentation must be by the rectum, and the lower bowel in this disease is generally in a fair condition for this method of nutrition. The subcutaneous or intravenous use of normal salt solution may be serviceable.

Courage and hopefulness on the part of the physician may do much good, and the services of a skillful and experienced nurse are of the utmost importance.

CHAPTER XVII

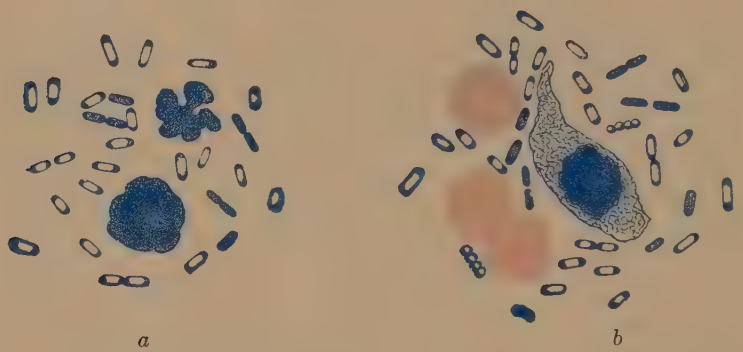
PLAGUE [1]

Ætiology.—The accounts which we have of the prevalence of plague date back almost as far as the earliest authentic records of history. As early as from the third and sixth century A.D. details have been furnished of widespread and devastating epidemics. The terrible ravages of the plague—the "black death"—in the fourteenth century are well known. Between 1346 and 1351 about twenty-five millions of people are said to have died of it in Europe, and about as many more in the Orient. Since then numerous epidemics have recurred again and again. In the last decades they have been limited chiefly to the Asiatic and African domiciles of the disease—above all, India, China, Persia, Egypt, etc. But the growing intercommunication of nations has always supplied opportunities for the spread of the disease, and within recent years not only America and Australia but also Europe has been threatened. But, thanks to our sanitary institutions, we need hardly fear it will ever again reach such dimensions in civilized countries as it did in previous centuries. The early and correct recognition and the thorough isolation of the first cases that spring up in a locality are of the utmost importance. Now that Yersin and Kitasato have identified the specific cause of the disease, the diagnosis has become simple and certain.

The plague bacilli (*vide* Fig. 21 *a* and *b*) are short, immotile rods readily stained in cover-glass preparation with anilin dyes, particularly with methylene blue. The ends of the rods take up the color much more strongly than do the middle portions (so-called "polar coloration"). The growing of a pure culture of the bacilli on ordinary nutrient media, too, offers no special difficulties. Permanent forms (spores) are not known. Warming to 60° C. (122° F.) kills the bacilli in a short time, boiling heat almost instantaneously. Dried on linen or the like, the bacilli may retain vitality for several weeks at mod-

[1] As I have had no personal experience with plague, the following presentation is based exclusively upon a study of recent literature on this important disease. I must mention, first of all, the monograph of H. F. Müller and R. Pöch in Nothnagel's "Specielle Pathologie." A good description of plague, as well as of all other infectious diseases of the tropics, is given in the commendable book of B. Scheube, "Die Krankheiten der warmen Länder." Jena, G. Fischer.

erate temperature. The plague bacilli occur in all specific manifestations of the disease, especially in the tissues and tissue juices of recent plague buboes, carbuncles, and skin vesicles. But in the pus of the spontaneous plague boils they are absent or only sparsely present. The plague bacilli are extraordinarily abundant in the sputum of plague pneumonia or in the terminal pulmonary œdema, as also in every drop of blood of severe, advanced cases.



a *b*

FIG. 21.—Plague bacilli. *a*, in pus; *b*, in sputum. (From Vierordt.)

The transmission of the disease proceeds at first from person to person. Chiefly through the expectoration, and less so through other excretions, the bacilli reach the patient's environment, and are then transmitted directly or through the mediation of fomites to another individual, by locating in a small skin lesion or by reaching and establishing themselves in the mucous membranes of the buccal and pharyngeal cavity, the nasal cavity or the bronchi. A sudden appearance of numerous cases, as occurs in some typhoid and cholera epidemics from infected drinking water, is not observable in plague. Usually plague epidemics spread but slowly, but ultimately they often attain remarkable dimensions. After the apparent cessation of the epidemic, there occurs not rarely a second outbreak, which under given circumstances may even be of greater violence.

An indubitably prominent rôle in the dissemination of plague is played by diseased animals, above all by rats. Like other rodents, rats are very susceptible to plague. The disease spreads among them to the greatest extent by their gnawing on dead animals, and as the excretions of rats contain plague bacilli, they surely contribute much to the spread from house to house. It has long been known that an outbreak of plague is often preceded by a striking mortality among rats. Insects (fleas, mosquitoes, etc.) may also contribute to the dissemination of the disease. [It is believed that fleas from infested rats, while biting human beings discharge excrement containing plague bacilli which are inoculated into the tiny wound by scratching. In the West squirrels have become infested with the plague.] From all that has been said, it follows that the more unfavorable the hygienic conditions, the more poverty, filth, and want exist in a community, the more easily does plague gain ground.

Symptoms and Course.—In developed cases plague usually begins quite suddenly with chills, high fever, and severe general symptoms. The latter consist in an unusually severe headache, in lumbar pains, and a feeling of very

great general debility. Very soon consciousness is clouded, a condition of mental stupor similar to inebriety supervenes, associated with complete apathy, or, in other cases, with conditions of anxiety and excitement. The fever is high (40° to 41° C. = 104° to 105.8° F.), remitting but little; the pulse is accelerated, at first full and dicrotic, and later small and irregular. The face is at first reddened and afterwards pale, the eyes sunken and fixed. The tongue is dry, and often looks as though whitewashed with lime. The spleen is distinctly swollen, and the liver, too, is enlarged. The urine is concentrated and generally contains albumen. There is moderate leucocytosis.

Under these manifestations of a severe constitutional infection, death occurs in some cases as early as two to three days (*pestis siderans*). As a rule, however, certain local affections especially characteristic of the plague accompany the general symptoms. According to their particular localization, the following three principal forms are differentiated:

1. *Glandular plague* (bubonic plague) probably develops more especially in those cases in which the bacilli have entered through small skin wounds and then spread along the lymphatic channels. On about the third or fourth day of sickness the lymphatic glands and the periglandular tissues in the inguinal region, in the upper femoral triangle, in the axillæ, in the neck, lower jaws, and elsewhere, according to the site of the infection, become greatly swollen. The buboes may grow to the size of a fist; in addition, the surrounding tissues also show inflammatory œdema. In the favorable cases the adenitis retrogresses, but, as a rule—if death does not ensue earlier because of the severity of the general infection—suppuration and rupture of the buboes occur. In these processes secondary infection with pus cocci (staphylococci and others) probably plays a prominent part.

2. *Dermal plague* is very much rarer than glandular plague. It is characterized by the appearance of "plague pustules" or "plague boils"—i. e., purulent vesicles—circumscribed hemorrhagic cutaneous necroses, and furunculous and carbunculous inflammations of the skin. In mild and favorable cases these inflammations, too, may become absorbed. But usually the advancing suppurative processes lead to ulcerative necrosis of the tissues.

3. *Pulmonary plague* is probably due to direct inhalation of plague bacilli. Under the clinical picture of a severe and often hemorrhagic pulmonary inflammation (plague pneumonia) it almost unexceptionally runs a lethal course. The presence of numerous plague bacilli in the expectoration has already been mentioned. This form of plague occurs more especially in persons already having pulmonary affections. Phthisical patients often die during a plague epidemic. Aside from this fact, many epidemics are distinguished by the frequent occurrence of pulmonary localization.

These three forms of plague show, naturally, manifold combinations and transitions, so that the total course of the disease may vary very much. Aside from the diversity of the localization, the severity of the infection also differs. There are mild and abortive types, severe and fully developed cases, and cases of the gravest infection with rapidly fatal termination ere the development of local manifestations can occur. (*Pestis siderans,* see *supra*). Complications with septic infections, meningitis, general hemorrhagic diathesis, etc., also take place. If the disease lasts for some time, marked remissions can often be seen on the third, sixth, and ninth days of the sickness. The malady

then seems to be composed of several relapses. Occasionally there may be an actual recurrence of the plague. Sequelæ of various kinds (paralysis, secondary inflammation) are but rarely observed.

Prognosis.—The prognosis of plague is very unfavorable. The mortality may reach fifty to sixty per cent, or even seventy to eighty per cent. Death usually occurs in the first six to eight days of the disease. If the disease takes a longer course, weeks may pass before convalescence begins, or death may occur even at a later period through general debility. We need not add anything regarding the pathologico-anatomical findings, as they are evident as regards their general features, at least, from what has been said of the clinical facts.

Prophylaxis.—The general hygienic and sanitary regulations for the combating of plague need not be entered into here. The efforts to find a method of prophylactic inoculation against plague (Haffkine) have so far yielded but little satisfactory results. Much cannot be expected in this direction, as even recovery from plague does not confer immunity against a second attack.

Treatment.—The treatment of plague must still be guided by the principles applying to all severe infectious diseases. We know no specific remedy, although modern experimental therapy is assiduously at work on the problem of producing an antitoxin. For the present, treatment consists chiefly in providing the best attainable general conditions (air, nourishment, location, etc.) and in giving due attention to the heart and respiration and the individual symptoms of the case. Regarding surgical procedures in plague buboes, plague carbuncles, etc., the opinions of experienced physicians are very divergent. Generally speaking, an expectant attitude seems more commendable than premature incision and extirpation.

CHAPTER XVIII

EPIDEMIC CEREBRO-SPINAL MENINGITIS

(Spotted Fever. Cerebro-spinal Fever)

Ætiology.—The epidemic form of cerebro-spinal meningitis has been known only since the beginning of the nineteenth century. The first epidemics were observed in southern France and in Geneva. Smaller ones occurred in Germany in 1822 and 1853; but it was not till 1863 that the disease became at all frequent among us. Since that date there have been more or less extensive epidemics almost every year in the southern and central portions of Germany. In 1904 and 1905 an unusually widespread and malignant epidemic occurred in upper Silesia. In 1906 and 1907 the disease showed itself especially in the Rhine Province.

Most of the epidemics appear in the winter and spring. We do not know any particular factors which promote the disease. It often seems to be decidedly endemic. Barracks, workhouses, and the like have been marked by tolerably extensive epidemics. The spreading of the disease to hitherto unaffected areas by patients is possible, but the danger is not very great (*vide infra*). The poorer classes, with their unfavorable housing and their lack of

cleanliness, are most exposed to the disease. Children and young adults are the most frequent victims, but now and then elderly persons are attacked. Sex cannot be shown to have much influence.

The direct cause of epidemic meningitis is the *Diplococcus intracellularis meningitidis* (meningococcus) first described by Weichselbaum. The meningococci are best found in the spinal fluid obtained by lumbar puncture; they lie in pairs, in shape like breakfast rolls, either in the leucocytes or alongside of them, and in appearance are very much like the gonococci. The meningococci have also been found in the blood and the fluid of affected joints. Regarding their existence in the nasal cavity, see below. They may be easily grown on Löffler's blood serum. They are of low vitality; desiccation, sunshine, cold, and the like readily kill them.

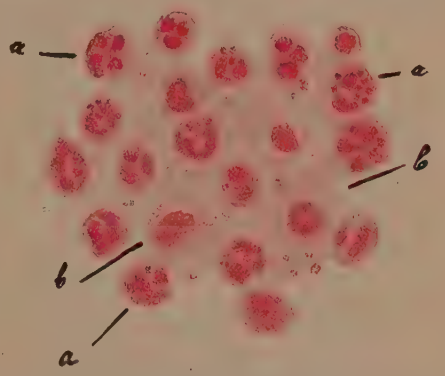


FIG. 22.—Meningococcus intracellularis in spinal fluid. *a*. polynuclear leucocytes with meningococci; *b*, endothelial cells.

Of fundamental importance for the question as to the kind of infection and the spread of the disease is the fact that the meningococcus is very frequently found in the secretion of the nose and the nasopharynx during the first days of the disease. The suspicion is therefore natural that the infection, as a rule, enters the body through the nostrils or the tonsils (pharyngeal or faucial). Whether they pass thence through the lymphatic channels directly into the meninges, or, what I consider more probable, only get there by way of the general blood stream, is not determined. The low vitality of the germ (*vide supra*) explains the fact that there is danger of severe visitation of the disease only where many persons are living together in close, damp quarters under unfavorable external conditions. In hospitals and in good apartments where sufficient caution is exercised the danger of contagion is very remote. A prominent rôle in the spread of the disease is, in all probability, played by the "cocci carriers." For it has been shown that many persons who are in contact with meningitis patients harbor meningococci in their nares without themselves being sick. Probably the transmission and spread of the disease occur to a greater measure through these healthy cocci carriers than through bedridden, isolated meningitis patients.

Not infrequently single sporadic cases of primary purulent meningitis or very small epidemics occur. The ætiology of these cases can be determined only by bacteriological examination. Occasionally, sporadic cases are also shown to belong to the epidemic form by the finding of the meningococcus. Often, however, sporadic cases are due to other infectious agents, especially the pneumococcus (*vide* the chapter on croupous pneumonia), the streptococcus, the so-called streptococcus mucosus, and others.

Pathological Anatomy.—The autopsy discloses an acute purulent cerebrospinal leptomeningitis. It is only in rapidly fatal cases that slight and incipient lesions have been met with. As a rule, the extent and intensity of the objective lesions correspond to the severity of the symptoms. In the brain

the purulent inflammation attacks the convexity as well as the base. It is usually most marked along the larger blood vessels and in the fissures of the cortex. Of the spinal cord the posterior surface suffers most. Frequently the lumbar portion is more affected than the parts above. It is, however, exceptional for the disease to be limited to the meninges; it is prone to extend into the underlying parenchyma. The microscope reveals clumps of pus corpuscles about the blood vessels, where they penetrate into the tissues, and not infrequently there are numerous centers of genuine encephalitis. These latter may be visible to the naked eye. Exceptionally there may even be cerebral abscesses of considerable size. The vessels are distended with blood, clear into the central ganglia, and ecchymoses are frequent. The cerebral ventricles are usually enlarged, and filled with a cloudy serum, or even with pus. It is plain that these lesions of the cerebro-spinal parenchyma greatly modify the clinical picture, and that they must frequently have more to do with the severity of the symptoms than has the leptomeningitis itself.

Clinical History.—Prodromata are relatively rare, and if present they are not severe, being confined to general malaise, with slight headache, and pain in the limbs. Usually the disease begins rather suddenly; there is intense headache, often felt mainly in the occiput, pain and stiffness in the back of the neck, and great general discomfort. It is not rare for vomiting to occur at first. Very often there are among the early symptoms such important mental disturbances as stupor or delirium. There is usually fever from the first. An initial rigor may occur, but it is not the rule.

The intensity of these first symptoms is not always the same. Subsequently to them the course of the disease may vary greatly. First there are very acute, violent forms, termed "explosive" (*meningitis cerebro-spinalis siderans, méningite foudroyante*), in which the cerebral symptoms are very severe, and the patient survives only a few days or even hours. Again, there are abortive cases, which also begin with apparently most dangerous, violent symptoms, but after a few days present a strikingly rapid and complete improvement. The majority of cases last about two to four weeks. In severe cases death may come as early as the first week. The disease is often protracted to six or eight weeks' duration, or even longer, and may end in death after all. Cases that last a good while sometimes exhibit a remarkably intermittent character. Improvement and relapses are very characteristic of meningitis. Finally, there are a considerable number of mild cases in which none of the symptoms is very pronounced, and recovery is relatively early.

The symptoms of the disease may be divided into (1) the severe general symptoms, referable to the brain and spinal cord; (2) the more localized, nervous symptoms; and (3) the results of the constitutional infection, including fever and symptoms in other parts of the body.

1. Among the less definite cerebral symptoms headache is important. It is usually terribly severe. It is chiefly occipital, but sometimes is frontal or temporal. Like most of the symptoms of meningitis, the headache undergoes very frequent changes in intensity during the course of the disease. For a time it may remit, only to recur with fresh severity. Marked vertigo and a sense of fullness in the head may accompany it.

The pain in the head is reënforced by intense pain in the nape of the neck and back, due to the spinal meningitis. There is almost always considerable

tenderness along the whole spinal column. The erector spinæ is contracted, making the back straight and rigid, or even producing opisthotonos; and the head is bent backward by the reflex contraction of the neighboring muscles. This has gained for the disease the German name "Genickstarre" (stiff-neck). (*Cf.* Fig. 23.) If an attempt be made at passive flexion of the head, violent pains at once ensue.

Fig. 23.—Severe case of cerebro-spinal meningitis. Stiff neck. General emaciation.

In most of the severe cases intelligence is blunted; we find all degrees of disturbance, from slight drowsiness to delirium on the one hand, or deep coma on the other. Some cases begin with marked maniacal excitement. These symptoms likewise may undergo frequent variation in their intensity. General convulsions occur in very severe cases alone, and are of evil omen.

The vomiting is also to be regarded as of cerebral origin. It frequently is an early symptom, but may be deferred.

2. Symptoms referable to the individual cerebral nerves are manifold and variable. The most frequent disturbances are in the nerves that supply the muscles of the eye. They include strabismus; nystagmus, or slow movements independent of volition; unilateral or bilateral ptosis; slow reaction of the pupils, or inequality of them, or myosis or mydriasis. In the area of distribution of the facial there is often a noticeable contraction of the muscles, giving the face a peculiar, painfully distorted look. Convulsive, irritative conditions in the muscles of mastication cause grinding of the teeth. Persistent trismus may occur, and is usually a bad sign.

Disturbance of the nerves of special sense is frequent. Deafness may be due to the stupor, but is often the result of an extension of the inflammation to the acoustic nerve. The purulent inflammation may be propagated as far as the labyrinth, or even into the middle ear. Tinnitus aurium is also frequent. Disturbances of vision are far less frequently observed, but optic neuritis has been repeatedly found by the ophthalmoscope. Severe metastatic ophthalmia and irido-choroiditis have been also observed. They are probably due to extension of the inflammation along the sheath of the optic nerve or to a hematogenous infection. Conjunctivitis sometimes occurs, but is probably caused by external injuries rendered possible by the imperfect closure of the lids, or the diminished sensitiveness of the parts. We have several times found the sense of smell diminished.

Of the disturbances in the area of distribution of the spinal nerves, the

most value in diagnosis is possessed by the cutaneous hyperæsthesia. It is apt to be particularly severe in the legs, and it may be so extreme that the light touch of a finger or a needle causes great pain. Sometimes there is a slight twitching in the muscles of the extremities. This has, however, no special significance. There are often rigidity and stiffness of the muscles. If the trunk of the patient be passively raised up in bed, the legs usually do not remain extended but are involuntarily flexed (Kernig's sign). As might be expected, there is no invariable rule about the reflexes. The cutaneous reflexes are usually well marked, and the tendon reflexes may be; but in some cases we have found the tendon reflexes markedly diminished or even abolished. Such a condition is probably due to some lesion of the fibers of the posterior nerve roots.

All of the nervous symptoms above enumerated result from one of two causes—either the roots of the nerves are affected by the purulent exudation, or the inflammation extends inward to the central organs themselves. This extension is the explanation also of other symptoms sometimes observed—viz., hemiplegia, paraplegia, partial convulsions, and aphasia.

3. In addition to all these nervous disturbances, we see also symptoms referable to other parts of the body. Of this class there is one cutaneous affection which is a very valuable aid to diagnosis. Herpes labialis or herpes facialis is apt to appear soon after the beginning of the meningitis. It is seen in more than half the cases, and as frequently in severe as in mild attacks. Other eruptions occur now and then—e. g., roseola, urticaria, or petechiæ. Sometimes they are so symmetrically distributed upon the two halves of the body as to suggest the idea of a nervous origin.

The digestive system seldom displays severe symptoms beyond the vomiting already mentioned. Anorexia and constipation are, indeed, usually present, as in many grave diseases. We have seen mild dysentery a few times. Now and then a slight jaundice has been noticed. The spleen is often somewhat swollen, but very rarely attains great size.

Swelling of the joints has been observed quite often; it is much more frequent in some epidemics than in others. The enlargement may be an early or a later symptom. It does not usually prove serious.

The urinary apparatus is seldom affected. The urine may contain some albumen and a few casts. Polyuria is an interesting symptom, probably of nervous origin. It is more apt to occur in the latter part of the disease. In a number of cases sugar has been found in the urine. Cystitis is a secondary disorder which is not very rare, particularly in severe cases when the catheter has been used.

Pulmonary and bronchial symptoms are likewise secondary. They occur very often in bad cases. It is evident how easily the stupor of the patient may lead to the inhalation of solid matter, with consequent bronchitis and lobular pneumonia.

Lesions of the circulatory system are rare. Acute endocarditis has been observed only a few times. The pulse is usually somewhat accelerated, seldom rendered slow. Very frequently the pulse rate is remarkably variable, undoubtedly because of variation in the supply of nervous force. Slight irregularities in the pulse are also common. In the blood we find quite a marked leucocytosis.

The *fever* in epidemic meningitis conforms to no single type. It does not correspond at all to the severity of the other symptoms; the worst cases may run their course with little or no fever. In most instances the fever has irregular remissions. It seldom exceeds 104° F. (40° C.). Sometimes the fever exhibits a decidedly intermittent character. It is in these cases particularly that we find the variation in the intensity of all the symptoms of which mention has been made repeatedly. The variations in the temperature do not, however, always run parallel with the changes in the other symptoms. In mild cases the fever is usually moderate and brief. The abortive attacks may present high temperatures at first, but these quickly abate. In case of a fatal issue there is sometimes hyperexia before death, reaching 108° to 109° F. (42° to 43° C.). In the severer but not fatal cases the fever declines slowly but irregularly. The fever may be over long before the other symptoms disappear.

In many cases there is exceedingly rapid and severe emaciation. The patients, especially children, become wasted to skeletons (Fig. 23). Undoubtedly the fever and defective assimilation of food are the greatest factors, but the simultaneous action of neurotrophic influences is undeniable.

It is impossible to portray all the forms, symptoms, and courses the disease may have. The chief varieties have been already mentioned; but in reality these are only types which run into one another without sharply defined border lines. It is in itself a characteristic of epidemic meningitis that most of the more tedious cases have a variable, uncertain course. We may even meet with a complete intermission of all the symptoms, lasting for quite a while, so that the return of the trouble may fairly be called a relapse.

Sequelæ.—Sequelæ are not rare after severe cases. Persistent deafness is most frequent. It results from the complications, already mentioned, which affect the labyrinth and the middle ear. Little children may become deaf and dumb. Again, vision may be deranged, because of retinitis, atrophy of the optic nerve, or corneal opacities, etc. It is not very rare for meningitis to leave grave nervous disorders behind it. These are frequently the symptoms of a chronic hydrocephalus. We may observe headache, sudden unconsciousness, or even convulsions, mental impairment, and weakness and ataxic uncertainty of the extremities. Or there may be localized disturbances due to permanent injury of limited portions of the brain or spinal cord, such as hemiplegia, paraplegia, and aphasia. From many of these conditions there may be a gradual recovery, but others prove incurable.

Diagnosis.—The diagnosis of cerebro-spinal meningitis is not difficult in a well-developed case, particularly if the prevalence of an epidemic puts us in mind of the disease. Diagnosis is more difficult in sporadic cases, and most difficult of all when the patient does not come under observation till he is very ill and when we cannot obtain the previous history. Important factors are the abrupt onset, the speedy appearance of grave cerebral symptoms, the characteristic headache and pain in the back, the stiffness of the neck, and the herpes labialis.

If we find evident symptoms of meningitis, we have still to decide whether the case is one of primary epidemic disease, or secondary, due, perhaps, to extension from some other part. Bearing this last possibility in mind, we should examine the ears carefully; for, as is well known, chronic otitis media

may set up a purulent meningitis. Again, it may be very difficult to exclude a tubercular meningitis. Here we should consider other circumstances that might render tuberculosis probable, such as the general condition of the patient, heredity, previous pleurisy, the results of thoracic examination, or scrofulous disease of the bones or joints. The existence of herpes points toward epidemic meningitis, for it is exceptional in the other forms of the disease. It is sometimes difficult to distinguish between meningitis and severe cases of other acute infectious diseases—e. g., typhoid fever and septic diseases. Here we must weigh all the circumstances carefully.

Lumbar puncture has greatly aided the diagnosis of meningitis. This was introduced by Quincke, and is now generally employed. As Quincke first showed, we can usually reach the subarachnoid space of the cauda equina without difficulty between the third and fourth lumbar vertebræ with an aspirating needle about 8 cm. long. The patient should lie on one side. In this way we can obtain more or less of any meningitic exudation that may be present.

In epidemic meningitis the spinal fluid is generally, though not always, distinctly cloudy, and deposits a more or less marked sediment of pus corpuscles, which, upon microscopical examination, are seen to be polynuclear leucocytes. As a rule, lymphocytes are not present, in contrast with tubercular meningitis. The bacteriological examination of the fluid with the demonstration of meningococci (*vide supra*) or tubercle bacilli, etc., is of decisive importance.

Prognosis.—The prognosis depends chiefly upon the severity of the cerebral symptoms. Yet we should be guarded in our utterances, even when the case seems mild, or has apparently made the first steps toward convalescence. The disease sometimes changes for the worse at a late period. In general the mortality is about thirty to forty per cent. Probably this estimate does not take into account many very mild cases. In the last extremely malignant epidemic in Upper Silesia the mortality rose even to sixty to seventy per cent.

Treatment.—In the last extensive epidemics many attempts have been made to find a specific treatment for meningitis. The transmission of meningitis to animals has not yet been successful. But the blood serum of animals previously injected with living or dead cultures agglutinates meningococci. Such sera have frequently been used for therapeutic purposes (Jochmann, Kolle and Wassermann, and others). The injections are given sometimes subcutaneously, sometimes after preparatory lumbar puncture, directly into the spinal canal. Definite curative results have not been obtained, and under these circumstances the treatment of epidemic meningitis must still be mainly symptomatic.

[Recent results are better. Of 400 cases treated by Flexner's serum, 295 recovered. Netter reported a mortality of only twenty per cent in the recent epidemic in Paris. At Evreux, of 18 soldiers treated with serum, only 2 died.]

A valuable remedy is cold applications. Ice bags are placed upon the head, and, if possible, along the spine. There are long and narrow rubber bags for the latter purpose. These applications are borne well by most patients and afford decided relief. The local abstraction of blood has also an undeniably beneficial influence, however difficult this may be to explain. Leeches are put behind the ears, and cupping glasses on the back of the neck and along the spine. Mercurial ointment is often rubbed in, not only locally but also in the same way as in treating syphilis. Its efficacy is doubtful. The narcotics are

of great value. The best is morphin given subcutaneously. It lessens the pain, and often affords the uneasy and delirious patient rest and sleep. Chloral, bromid of potassium, antipyrin, sodium salicylate, etc., may also be employed. Iodid of potassium is often given internally, to the amount of 22.5 to 30 gr. (gm. 1.5 to 2) in a day, especially in cases with a slow course. Iodid of sodium has been particularly recommended in daily amounts of 15 to 22.5 gr. (gm. 1 to 1.5) in solution. [Urotropin has recently been recommended.] The evacuation of a part of the meningitic exudation by lumbar puncture (*vide supra*) is sometimes followed by a temporary improvement of the symptoms. Some physicians earnestly advocate lumbar puncture repeated every four or five days. The author can corroborate the favorable symptomatic influence of lumbar puncture in some, though not all, cases, but, after the experiences of the last great epidemic, must warn against exaggerated expectations.

The fever hardly ever requires special treatment. Quinin exerts no influence in the intermittent type. Antipyrin is better borne, and it also, like the other nervines, sometimes relieves the nervous symptoms. Cool baths are not to be recommended, but warm and hot baths or hot packs sometimes seem to be of decided benefit. Local complications—e. g., affecting the eye or the ear—require special treatment. The swelling of the joints which sometimes occurs we have thought to be somewhat relieved by salicylates, aspirin, etc.

CHAPTER XIX

SEPTIC AND PYÆMIC DISEASES

(Spontaneous Septico-pyæmia)

The septic and pyæmic processes which follow serious injuries or operations belong to surgery; but analogous diseases occur in persons who are apparently in perfect condition. They take the form of an extremely severe acute infectious disease, usually fatal. There can scarcely be a doubt that in almost all these cases there is some small break in the continuity of the external skin or the mucous membrane which affords an entrance for the infectious material; but, since the infection itself is wholly unnoticed, the general disease seems a primary affection, and its correct interpretation often occasions the physician considerable difficulty. Even if the septic infection be correctly recognized, it is sometimes impossible to determine the point of origin. For such cases Leube has introduced the name of "cryptogenic" septico-pyæmia. We must add, however, that, in many cases, at least, a careful inquiry and examination will reveal the origin of the infection. In many cases, however, a complete explanation of the occurrence of the infection is given only by the autopsy, and the origin of some cases remains obscure even after a careful autopsy. Not infrequently a general septic infection follows some other disease, as scarlatina, diphtheria, or one such as erysipelas or furunculosis, which, though itself due to pus cocci, is at first localized. In such cases we speak of a secondary sepsis.

Ætiology.—The causes of pyæmic and septic affections are chiefly the same pus cocci which produce many circumscribed inflammations and suppurations, especially the *Streptococcus pyogenes,* the *Staphylococcus pyogenes albus,* and the *Staphylococcus pyogenes aureus.* But *pneumococci, B. coli, gonococci, meningococci, B. pyocyaneus, proteus,* and other varieties may also lead to a general septic infection. A clinical differentiation of septic affections, according to the special kind of disease produced, is not strictly practicable. But, in general, it may be said that the graver cases of septic infection with extreme constitutional intoxication, hemorrhage, bacterial foci, etc., but without real multiple suppurations, are due especially to the streptococci. The staphylococci show a decided tendency to form metastatic abscesses. Following the old usage, cases with multiple pus foci are termed pyæmia.

It is very important for the correct understanding of all these conditions to bear in mind that, in different cases, different organs or parts of the body may become the chief seat for the localization of the micrococci. Hence severe local disorders may arise, which, of course, may give diverse aspects to the clinical picture according to the special localization. This is the reason why many of these affections were formerly regarded as separate diseases, although in reality they were only different localizations and forms of the same infectious process. Among these affections were the so-called "acute osteomyelitis" almost always caused by the *Staphylococcus pyogenes aureus,* "malignant endocarditis," certain forms of "malignant erysipelas," etc. In their clinical aspect these differences are, of course, still of great importance, but the ætiological connection of all these cases must always be emphasized, because only by putting a proper stress upon this point can we obtain a correct understanding of all the combinations manifested in the clinical course of the disease.

Before we consider the details of the anatomical changes in septic affections, we would mention those circumstances (exciting causes) which, we know by experience, most commonly cause or render possible the occurrence of septic infection. The following are chiefly to be considered: 1. The puerperal processes take the first place. Both after delivery, and still more frequently after abortion, the raw surface of the uterus may be the door of entrance for the septic poison. Artificial, criminal abortion, often performed with great carelessness, not rarely leads to septic infection. The physician must, therefore, always bear this possibility in mind. A gross pathological change is not necessarily to be seen in the uterus itself or in its appendages. We do find, often enough, diphtheritic and gangrenous inflammation at the place where the placenta was inserted, or purulent thrombi in the veins of the uterus and of the pelvis, etc.; but in other cases the uterus is merely a gate of entrance for the poison, remaining itself normal. 2. The septic poison may also be absorbed through slight abrasions of the skin, injuries, felons, whitlows, etc.; and these may be almost completely healed by the time the severe symptoms of disease are developed. Bedsores belong in this category. 3. Ulcers of the mucous membranes may give rise to infection. Septic infections, unfortunately, not infrequently develop from sore throats of all kinds. At other times the infection starts from the nose or ear (middle-ear abscess). The most frequent intestinal causes are ulcerative proc-

esses in the vermiform appendix and the many forms of cholecystitis (usually through *B. coli*). From the urethral mucosa a gonococcus sepsis infrequently develops. Often the urinary passages give rise to an infection with B. coli or staphylococci. 4. A peculiar position among septic diseases is held by acute endocarditis, which often develops on the basis of an old chronic endocarditis. This form of sepsis will be specially discussed later on. 5. Lastly, we sometimes find no other source for general sepsis than a suppurating disease of the bones, joints, or other parts, previously existing. We must here suppose that some connection was formed between the original pus cavity and the blood or lymph channels, and that thus the micrococci entered the general circulation and were enabled rapidly to increase.

Pathological Anatomy.—The most striking feature at the autopsy of such cases is that there is never found a lesion of one organ exclusively. Several, or it may be almost all, of the organs exhibit numerous limited foci of disease. The lesions sometimes consist for the most part of multiple abscesses, sometimes of numerous ecchymoses, and sometimes of combinations of the two. The abscesses are found chiefly in the lungs, kidneys, liver, spleen, muscles, heart, brain, and thyroid gland. Quite extensive purulent inflammation is also found. This attacks the joints or causes phlegmon of the muscles or skin by preference, but it also attacks the pleura, the meninges, and the eye, where it causes purulent choroiditis, panophthalmitis, and purulent degeneration of the vitreous. Purulent phlebitis also occurs. The ecchymoses are most frequent upon the surface of the body, the serous membranes (pericardium, pleura), the retina, the conjunctiva, the brain, the pelvis of the kidney, etc. Besides these multiple abscesses and ecchymoses, there is frequently another disorder, which seems to be the very focus of the disease, viz., acute ulcerative endocarditis (*cf.* the appropriate chapter). This usually attacks the mitral valve, more rarely the valves of the aorta, and quite exceptionally the valves of the right side of the heart. Finally come a number of changes common to all severe constitutional infectious diseases—acute splenic tumor, "cloudy swelling" of the liver and kidneys, a dryness and dark-red color of the muscles, etc.

In regard to the special origin of all these symptoms we may certainly refer the abscesses, the purulent inflammation, and the endocarditis to the immediate presence of the micrococci themselves, while the parenchymatous degenerations of different organs, the hemorrhages, and probably the splenic enlargement and the diffuse acute nephritis, are to be regarded as due to toxic action. Microscopic examination of the internal organs gives us a very instructive insight into the morbid process. This often reveals the presence of many tiny foci of disease, in the center of which we often see a little blood vessel completely filled with micrococci ("micrococcus embolus"). As Weigert first found, the primary action of a little focus of micrococci upon the surrounding tissues consists of a circumscribed cell necrosis, a small focus of "coagulation-necrotic" cells without nuclei. This may be followed later by circumscribed suppuration.

Clinical History.—It is our intention to discuss below those cases chiefly which are of interest to the physician rather than the surgeon—i. e., where the septico-pyæmia is an apparently primary, acute, and grave disease. Many of the essential traits of this type of disease are identical with those of the pyæmia

which complicates the effects of serious wounds or the inflammation subsequent to childbirth. But it is precisely the apparent absence of a cause which renders many cases of this form of sepsis so obscure and difficult to understand. Besides, the patient is often in a stuporous condition when the physician is called; and this adds greatly to the difficulties of a correct judgment.

The beginning of the disease is usually rather abrupt. An apparently healthy person is attacked with febrile symptoms, headache, and "rheumatic" pains in the muscles, joints, and loins. There may also be gastro-intestinal symptoms of considerable severity, including vomiting and diarrhea. Usually the patient feels ill enough to take speedily to his bed. The symptoms now increase rapidly, and develop into a severe illness which may resemble either a bad case of typhoid fever or miliary tuberculosis. Or the cerebral symptoms, such as headache, stupor, and delirium, may become so prominent that the attack seems like meningitis. If the trouble in the joints (*vide infra*) predominates and there are signs of endocarditis, the disease may at first be taken for a violent attack of acute articular rheumatism.

Taking up the separate symptoms, we shall first name those which belong to every severe acute infectious disease and have nothing characteristic about them. In this list belong the general prostration, the anorexia, the mental disturbance, the stupor and delirium, the headache, the subjective symptoms of fever, the dryness of the tongue, and finally the acute splenic tumor which can often be made out. There are, however, other and more characteristic symptoms; and it is chiefly upon these that the diagnosis rests, provided we can make one at all. These are:

1. *The Course of the Fever.*—In many cases, it must be confessed, this is not at all characteristic. It may even be so like that of typhoid fever, at

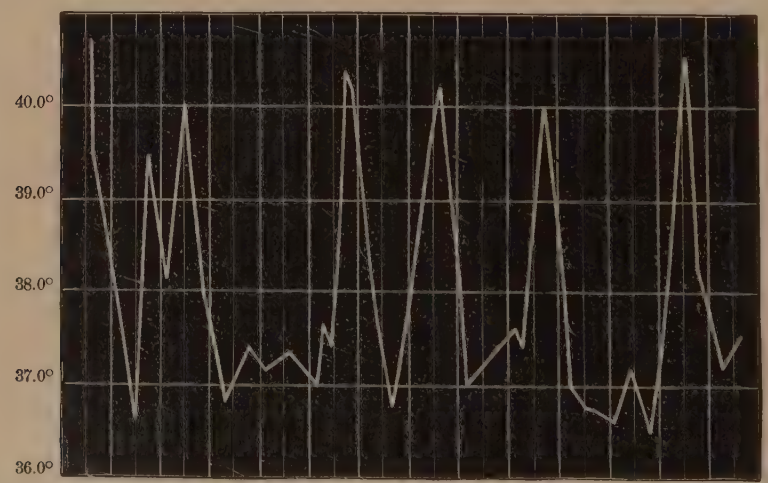


FIG. 24.—Fever curve in pyæmic infection. The marked elevations in temperature were always ushered in by a chill. (Erlangen Medical Clinic.)

least for some time, as to lead to a wrong diagnosis. In other cases, however, the temperature curve does aid us greatly—viz., when it represents an intermitting fever with marked elevations, reaching 106° F. (41° C.) and higher,

and often accompanied by a chill, and with subsequent deep depressions. The curve may thus come to resemble closely that of a quotidian or even tertian intermittent fever. This true "pyæmic" fever, in which the rise in temperature is usually associated with a severe chill, occurs chiefly in the cases with multiple abscesses. Sometimes, again, the course of the fever is made up of similar paroxysmal elevations, separated by periods of ordinary remitting fever.

Generally streptococcus infections have a more irregularly remittent or, in severe cases, an almost continuous fever. Intermittent attacks of fever occur especially in infections with staphylococci, bacteria coli, and gonococci.

2. *Cutaneous Symptoms.*—These are very frequent, and a great aid to diagnosis. The hemorrhages into the skin are of chief importance. They may be either punctiform petechiæ or more extensive ecchymoses. Of other cutaneous appearances, the first in relative frequency is an erythema resembling scarlatina. It is not improbable, as we have already said, that many cases which have been described as severe scarlet fever occurring during childbed were in reality septic disease. We also sometimes see eruptions resembling erythema exudativum multiforme or erythema nodosum, and also in some cases roseola, pustular eruptions, and herpes. Extensive inflammations of the skin, like erysipelas, are especially characteristic of certain cases. They are seen on the lateral surfaces of the thorax, the back, the thighs, etc., and often end in phlegmonous suppuration. When they occur we can usually make a very positive diagnosis of septic infection.

3. *Circulatory Disturbances.*—An ability to recognize the cardiac lesions would be very desirable; but often this is impossible before death. Endocardial murmurs are often wanting, even in cases where the autopsy discloses abundant exudation and ulcers upon the valves. Still, in some cases of this sort we have found the heart sounds noticeably deficient in clearness. Sometimes we hear very loud or low, blowing sounds. The cardiac dullness is often quite normal, in other cases somewhat increased. In some cases fibrinous or purulent pericarditis develops. Functional disturbances of the heart's action are almost always present. The heart's action is excited and very rapid (120 to 140 or more), or in rare cases abnormally slow. Irregularity and inequality of the pulse are common. The tension of the pulse is usually low, and the frequent pallor and mild cyanosis of the patient point to a diminished energy in the heart's action and impaired vascular tone. All these disturbances are due partly to the effect of the toxins, and partly to an acute (circumscribed) myocarditis. In the blood there is, almost without exception, more or less marked leucocytosis and a diminution in the number of red corpuscles, which in the severe cases may lead to a most profound anæmia. The demonstration of the pathogenic organisms in the blood is of the greatest importance. (*Vide infra,* "Diagnosis.")

4. *Cerebral Symptoms.*—The grave cerebral symptoms are for the most part quite analogous to those of other severe acute infectious diseases. They may be present, and yet no marked objective cerebral lesions may be found after death. In other cases they have an anatomical basis—in purulent meningitis, hemorrhagic pachymeningitis, cerebral hemorrhage, or abscess. These conditions, just enumerated, may excite focal cerebral symptoms—e. g., hemiplegia.

5. *Affections of the Joints.*—Affections of the joints are comparatively frequent, and of great value in diagnosis. We may find serous, or more likely purulent inflammation, or even peri-articular abscesses. If they appear early in the attack, they may, as we have said, lead to an erroneous diagnosis of acute articular rheumatism. Suppurative processes affecting the periosteum and the marrow of the bones not infrequently accompany the joint affections. Only in rare cases do the bones remain wholly unaffected, as is evident from the frequent occurrence of pain in the long bones. If there is decided suppuration in the bones we speak of an acute osteomyelitis. This is seen especially in the lower extremities. It is almost always occasioned by the *Staphylococcus aureus.* In earlier times such cases were termed bone-typhoid. Finally, abscesses and extensive phlegmonous suppuration in the muscles are not uncommon.

6. *Renal Changes.*—Renal changes are very frequent, but abscesses and hemorrhages may be numerous in the kidneys, or hemorrhages in the mucous membrane of the pelvis of the kidney, without materially altering the character of the urine. In most cases, however, an acute septic nephritis is conjoined with the infarctions and abscesses, and then the urine exhibits all the characteristics of acute Bright's disease, having a small or large amount of albumen, red and white blood corpuscles, epithelium, and casts.

7. *Pulmonary Symptoms.*—The pulmonary symptoms are in part secondary. Bronchitis and lobular pneumonia develop as in all other severe constitutional diseases. Embolic foci of pneumonia and the pulmonary abscesses, as such, rarely produce distinct objective physical symptoms, but frequently there is a striking dyspnœa which is out of proportion to the insignificance of the physical symptoms. Empyema is a not infrequent result of infection of the pleura, due to the foci of disease which are situated upon the outer surface of the lungs, or perhaps sometimes to the condition of the blood. If the aspirating needle shows the actual existence of empyema, this fact may make the diagnosis of the constitutional disease much easier. A fibrinous pleurisy of slight degree is often found at the autopsy, and it may be diagnosticated during life by the detection of a slight pleuritic friction rub.

8. *Abdominal Symptoms.*—Of the abdominal symptoms we have already mentioned the acute splenic tumor. It is almost impossible to diagnosticate infarctions and abscesses in the spleen. If the spleen is enlarged and noticeably painful, we may suspect their existence. There are sometimes quite severe intestinal symptoms, such as a profuse "septic diarrhea," in cases where the autopsy does not show any particularly grave lesions. Still, intestinal ecchymoses and intestinal diphtheria have sometimes been observed. We should mention that often the skin has a faint jaundiced hue. This is sometimes the result of duodenal catarrh, but more frequently it is to be regarded as a "hepato-hematogenous" jaundice due to a decomposition of the blood.

9. *Ocular Disturbances.*—The purulent inflammations of the eye, which are probably of embolic origin and which may develop into diffuse septic panophthalmitis, have been known for some time. Lately, Litten and others have called attention to more minute changes in the fundus of the eye. These are revealed through the ophthalmoscope, and have great diagnostic value. Chief among them is retinal hemorrhage, which is sometimes accompanied by a white

spot in the center, corresponding to a necrosis of the retina in that place; but there may be similar white spots without hemorrhage.

Course of the Disease and Prognosis.—In severe general septic infection, death sometimes occurs in a few days. In other cases the disease continues longer, the symptoms lasting two or three weeks or even more. Improvement sometimes takes place, followed by a relapse. Here, too, the result is often fatal, although the infection may finally be completely overcome. We may also accept as certain that there are mild, curable forms of septic disease. In these the signs of general infection may predominate, such as fever with general intoxication, cardiac weakness, pains in the joints, albuminuria or exanthemata, or, in other cases, the chief symptoms may be due to a special localization of the germs or their toxins (acute endocarditis, septic nephritis, septic inflammation of the serous membranes, septic enteritis, etc.). As we shall see later, the so-called hemorrhagic diseases, as also acute articular rheumatism, acute endocarditis, erythema exudativum, and other diseases have many close connections with the milder forms of septic infections.

Diagnosis.—It is self-evident that a disease which combines symptoms so manifold and so ambiguous must be very difficult to recognize. Especially in the prebacteriological times was the diagnosis of sepsis often impossible. To-day the advance in bacteriological blood examination has very greatly perfected our diagnostic technic for acute infectious diseases. On the basis of bacteriological blood findings, it is not only possible in most cases to make a diagnosis of septicæmia, but also to determine the special kind of infectious agent (streptococci, staphylococci, B. coli, pneumonococci, etc.). None the less, for obvious reasons, this method of diagnosis cannot at present be generally employed in medical practice, and therefore the purely clinical diagnostic means, though often far less certain in their results, still retain their value. We will recapitulate the chief diseases to be excluded. A case may greatly resemble typhoid fever when there is persistent prostration, diarrhea, an eruption like roseola, and an enlarged spleen. In discriminating, we should consider with great care the possible ætiology—e. g., external injuries, etc.; we should remember that most septic affections begin rather more abruptly; and we should look for swelling of the joints, cutaneous ecchymoses, nephritis, phlegmonous suppurations, an intermitting form of fever, and septic disease of the retina. Leucocytosis is a very valuable sign, since it is almost always absent in typhoid fever, but a positive result from Widal's serum reaction renders a diagnosis of typhoid very probable. It is all the more possible for the disease to resemble meningitis, because, as we have said, meningeal disturbance may be one of the symptoms of the sepsis and color the whole picture. Here the characteristic symptoms of septic poisoning already mentioned would be of some value in diagnosis, and the physical signs of endocarditis or of a greatly enlarged spleen would be worth still more. The final decision will often depend upon the result of lumbar puncture. There may be much difficulty in the differential diagnosis between acute sepsis and acute miliary tuberculosis. Here we should consider carefully each separate symptom, and, above all, the ætiology, searching for something that would explain the occurrence of sepsis on the one hand, or of acute miliary tuberculosis (*q. v.*) on the other. If we found miliary tubercles in the choroid by means of the ophthalmoscope, or tubercle bacilli in the sputum or in the blood (which is, of course, not always

possible), all doubt would vanish. A marked diazo-reaction of the urine, too, argues for acute tuberculosis. At the beginning of a septic attack the rigors may arouse suspicions of intermittent fever. Usually the early appearance of other symptoms corrects this idea; but, if not, the powerlessness of quinin will. If a severe acute nephritis has developed itself in a septic case, all the symptoms may be erroneously referred to uræmia; but persistent observation will usually lead us to the right conclusion. As to the conditions of great prostration resembling acute sepsis, which occur in acute (primary) ulcerative endocarditis and in severe articular rheumatism, see the appropriate chapters.

In general, the diagnosis of acute sepsis and septico-pyæmia can rarely be settled during the first days of the disease, but on further observation and with due attention a tolerably definite diagnosis can usually be made. The chief elements for diagnosis are a careful consideration of the ætiology (previous circumscribed suppurations, sore throat, etc.) and attention to the whole clinical picture, as well as to any individual symptoms especially characteristic of septic infection (purulent inflammation chills and fever, cutaneous and retinal hemorrhages, nephritis, joint swellings, splenic tumor, pale, icteric complexion, marked leucocytosis, etc.).

If there be persistent high intermittent fever with chills, without any demonstrable local affection and an often fairly good general condition for a long time, we must suspect a hidden pus focus in the body. This point must always be carefully borne in mind because of the possibility of surgical treatment. Examine most thoroughly and give due attention to the subjective complaints of the patient (pain, sensitiveness to pressure, etc.). Concealed suppurative processes in the abdominal cavity (subphrenic or perinephritic abscesses) are especially apt to be overlooked.

Treatment.—The treatment, aside from cases in which surgical intervention is indicated, is merely symptomatic. Of course we try again and again to cut short the attacks of fever by large doses of quinin, antipyrin, pyramidon, etc., but never with lasting success. With marked swelling of the joints we may try the salicylates. Of other remedies, cardiac stimulants (alcohol, camphor, caffein) are most frequently employed, and narcotics if necessary. At times the repeated subcutaneous infusion of sterile physiological saline solution is most appropriate, especially when cardiac strength is failing and vascular tension lessening. Great stress is to be laid on keeping up the physical strength by careful nourishment. Specific treatment, promising as its future may be, has not yet attained positive success. Others and we ourselves have not seen indubitable results from the various antistreptococcic sera, any more than from the chemical preparations recommended (argentum colloidale named "collargol," twenty-five per cent iodipin subcutaneously, 5 to 10 c.c. = ℥lxxv to ʒijss.). None the less, these remedies will frequently be resorted to in practice.

Circumscribed suppurative processes, which may develop (*vide supra*), of course demand appropriate surgical treatment (empyema, phlegmons, deep-seated abscesses, etc.). The recent efforts to treat puerperal fever and other septico-pyæmic infections arising from the uterus by tying off the afferent veins, can only be briefly mentioned here. [What is said of specific sera applies also to vaccinations with sterilized bacteria, preferably obtained by blood

culture. In an acute and severe case of sepsis there is scarcely any hope of their doing good; in a milder and more chronic infection the possibility of benefit is a trifle greater.]

CHAPTER XX

TETANUS

(Lockjaw)

Tetanus is an acute infectious disease whose chief symptom is the occurrence of severe, extensive, tonic ("tetanic") muscular spasms. The specific cause of tetanus, the tetanus bacillus, was first discovered by Nicolaier in garden earth, and examined more closely in pure culture by Kitasato. Rosenbach first cultivated the same bacilli from the secretion of the wound of a man who died of traumatic tetanus. Since then the specific significance of the tetanus bacillus has been positively confirmed beyond any doubt by numerous investigations. These bacilli are slender motile rods, distinguished by a little head spore at one end (*vide* Fig. 25). These spores are remarkable for their resistance to external influences (heat and desiccation). The tetanus bacilli grow only anaërobically—i. e., under the greatest possible exclusion of oxygen. Their growth is therefore facilitated by the presence of other bacilli which consume oxygen. If a small quantity of bacilli is injected under the skin of mice, rabbits, or guinea pigs, the animals die under the most violent tetanic spasms in from twenty-four to thirty-six hours. Since the development of the bacteria remains limited to the seat of the wound or the injection, it is *a priori* probable that the spasms are not excited immediately by the bacilli, but by a chemical poison produced by them during life. In fact, Brieger has lately succeeded in producing several alkaloid-like substances from tetanus cultures, so-called toxins, which he terms tetanin, tetanotoxin, and spasmotoxin. All these substances are violent poisons, and, like strychnin, provoke the most violent tetanic spasms in the animals experimented on.

Fig. 25.—Tetanus bacilli.

According to the investigations of H. Meyers, the tetanus toxins are first absorbed by the peripheral nerves and thence migrate up to the central organs.

In regard to the method of infection in man, it occurs by far most frequently through open wounds (operation wounds, or small accidental injuries). Since the tetanus bacilli are found chiefly in the soil, we can readily understand that injuries of the feet in persons who go barefoot, or injuries of the hands in gardeners, farmers, etc., most frequently give rise to tetanus. In view of the wide prevalence of the tetanus bacilli, the rareness of tetanus in human beings is striking. This is probably explained by the anaërobic requirement for growth. Therefore, the most danger exists in somewhat deep wounds, situated beneath the skin, that are rendered unclean by garden earth (stab wounds [blank-cartridge wounds], long wooden splinters, etc.). The fact is

of practical importance that the tetanus bacilli are not infrequently found in the cæcum of herbivorous animals (horses and cows). Therefore, earth mixed with dung is especially infectious. Horses frequently suffer from tetanus. Formerly, in place of traumatic tetanus, the term idiopathic tetanus was employed where no external injury could be detected as a point of infection. [Commercial gelatin may contain tetanus bacilli, and therefore should be thoroughly sterilized before it is used for therapeutic injections for bleeding.] Aside from small, unnoticed external injuries, infection may take place through the mucous membranes (pharynx). If, as is often the case, the affection is preceded by a severe cold or wetting, we speak of a rheumatic tetanus. *Tetanus neonatorum* is undoubtedly an ordinary wound tetanus, almost always arising from an infection of the umbilicus.

In Germany [and temperate latitudes generally] tetanus is a comparatively rare disease. Men are much oftener attacked than women. Tetanus is much more common in the tropics than in our climate. The frequency of the disease in negroes is well known. Tetanus is also not equally frequent at all times. Endemics and epidemics of tetanus have often been observed, especially in time of war. These have arisen in part from the unfavorable influence of certain external conditions, such as poor care, bad weather, sleeping on the wet ground, etc.

Clinical History.—In so-called "rheumatic" tetanus the symptoms usually begin soon after exposure to the exciting cause. There may be, however, an interval during which the patient feels perfectly well, or at most has only certain mild and indefinite premonitory symptoms, such as languor and headache. Similar prodromata may occur in the apparently idiopathic cases.

Traumatic tetanus seldom begins immediately after the injury has been received. Several days or even weeks may intervene previous to the outbreak of the disease. Here, too, there may be mild prodromata for a brief period preceding the graver phenomena. The patient's wound presents, as a rule, no specific appearances. Tetanus may be associated with either slight or severe injuries, whether treated carelessly or kept apparently aseptic.

The symptoms of the disease proper are the same in both rheumatic and traumatic tetanus. They usually begin gradually. Ordinarily, the first thing noticed is a feeling of rigidity and tension in the muscles of the face, lower jaw, and back of the neck. The stiffness spreads by degrees to the muscles of the back and abdomen. The disease is sometimes completely developed in a few hours, but sometimes not till after several days.

The persistent tension of the facial muscles gives the countenance a strange immobility (*vide* Fig. 26). The brow is usually wrinkled, and the corners of the mouth are often drawn back in a "sardonic grin," or the face has a tearful expression from the deepening of the nasolabial folds and the drawing down of the corners of the mouth. Most prominent of all is the tonic spasm of the masseters, or trismus. The teeth are so firmly pressed together that it finally becomes impossible to open the mouth more than a few millimeters. The eyes are staring, the pupils usually contracted. The muscles at the back of the neck draw the head somewhat backward, but in many cases it can still be moved quite well, although in other cases it is fixed by the spasm. The spinal column is bent forward, so that the trunk is convex anteriorly, permitting the hand to be passed between it and the bed—opisthotonos. The muscles

of the back are hard and contracted. The epigastrium and the anterior part of the abdomen are flat. The abdominal muscles are as hard as a board. The lower limbs may be rigidly extended at the knee, and the adductors are also contracted, but, as a rule, the feet and toes are free from spasm. The arms

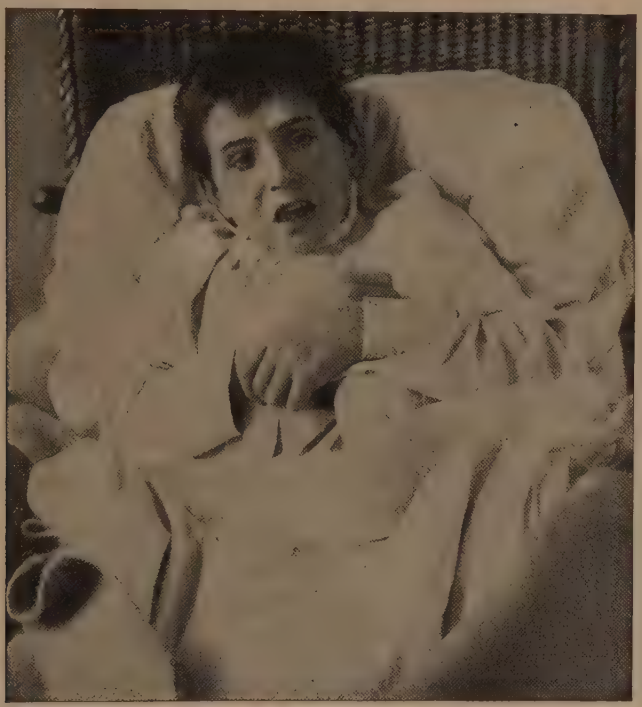


FIG. 26.—Facies in tetanus. The mouth cannot be opened wider on account of the existing trismus (personal observation).

generally can be quite freely moved, but the movements at the shoulder are usually decidedly impaired. Convulsive dysphagia, as seen in hydrophobia, may occur, but it is rare (*vide infra*).

In many cases the continuous tonic spasm is occasionally interrupted by sudden paroxysms, during which all the affected muscles become still more tense. In severe cases this gives the whole body a violent shock, and makes the opisthotonos even more pronounced. In a very bad case the paroxysms are very frequent; in a mild case they are rare or almost indistinguishable. Sometimes they are apparently spontaneous, and sometimes they are evidently of reflex origin, being superinduced by external irritation, often quite insignificant, such as a slight jar of the body, noises, etc.

As to other nervous derangements, little is known about them—partly, no doubt, because it is seldom possible to make an extended examination of the patient. Sensation is said to have been impaired in some instances, but in others it is perfectly normal. The muscles affected by the spasms are usually the seat of severe pain. The cutaneous reflexes are almost always exaggerated. In several cases which we saw very recently, the patellar reflex was much

increased, and in one there was distinct ankle clonus. Paralysis is extremely exceptional. There is often profuse perspiration. The forehead is usually studded with many beads of sweat. The intellect remains perfectly unclouded.

There is a special form of tetanus which must be briefly mentioned. It was first described by E. Rose, and is called "hydrophobic tetanus," or "cephalic tetanus." It occurs only in connection with injuries situated in the distribution of the cranial nerves—that is, in the face and head—and is characterized in most cases by violent spasm of the muscles of the pharynx. This is in addition to the other ordinary phenomena of tetanus. The disease in many ways reminds one of hydrophobia. Another characteristic point is that in most cases there is facial paralysis on the injured side.

Tetanus, as a rule, gives rise to no disturbances referable to the internal viscera. In one case, however, in the Leipsic hospital, croupous pneumonia and acute nephritis came on a few days before the end. Often there are dyspnœa and a most harassing sense of thoracic oppression—symptoms due mainly to the convulsive rigidity of the muscles, by which the thorax is constantly maintained in the position it normally assumes during inspiration. Expectoration is impeded; and, finally, there may be such an accumulation of secretions in the mouth and air-passages as to cause a secondary diffuse bronchitis, or an inhalation pneumonia. Another occasional source of extreme dyspnœa is spasm of the glottis.

The pulse often remains normal for a long while, but it is usually accelerated, not infrequently reaching 120 or 160 beats a minute in severe cases. Such a pulse is small, and may be somewhat irregular. The temperature is at first usually normal, or slightly elevated (100° to 102° F. [38° to 39° C.]). Later it is almost sure to rise, and, as Wunderlich pointed out, it is often very high shortly before death—for instance, 107° to 111° F. (42° to 44° C.). It is not rare for the temperature to keep on rising for a short time after death. No explanation of this terminal elevation of temperature has yet been furnished. It cannot be the result of the increased production of heat occasioned by the muscular spasm, for in earlier stages the most violent convulsions are unattended by any such change. Authorities are, therefore, inclined to assume that at the last there is a paralysis of the centers which regulate the warmth of the body, just as is seen in other severe nervous disorders, such as meningitis, injury to the cervical portion of the cord, and uræmia.

Interesting observations have been made with regard to tissue-metamorphosis during tetanus. The excretion of urea is not increased. This fact agrees well with Voit's view, that muscular activity has no relation to the breaking down of albuminoids. Senator failed to find any increase of kreatin and kreatinin in the urine. Probably the production of carbonic dioxid is abnormally large in tetanus. At least, physiological considerations would strongly indicate this, although it has not yet been actually demonstrated. Occasionally traces of albumen and sugar have been detected in the urine. There is usually obstinate constipation, probably due to the persistent rigidity of the abdominal muscles; and, indeed, micturition is not a little impeded from the same cause.

It may be said, in regard to the general course of the disease, that there are severe and mild forms of the disease. What has been said above applies mainly to the severe form. In this, all the symptoms reach their extreme violence in a

few days, the paroxysms occur in quick succession, and death usually takes place within a week or two. The fatal result is brought about by the suspension of respiration and by cardiac failure. Of course, the extreme difficulty of taking an adequate amount of food has an unfavorable influence. The bad cases seldom outlast the first week. If they do, there is some slight hope of recovery; the paroxysms may gradually become less frequent and less severe, until they finally cease altogether. The severer form, however, so rarely ends favorably that the prognosis is always very grave. The mild form, on the contrary, usually runs a much more favorable course. In it, all the symptoms are from the first much less severe. Often there is only more or less trismus, accompanied by no marked spasm in the muscles of the trunk, if any at all. There is little constitutional disturbance. The temperature is normal, and the prognosis is rather favorable. The disease may sometimes drag on for some weeks, but it often ends in complete recovery. It must not be forgotten, however, that what at first seems a mild case may develop into the severe form.

The anatomical changes in the nervous system in fatal cases are almost wholly negative. Further investigations are necessary to determine how far the finer changes in the motor cells of the spinal cord, which have repeatedly been found of late, are of pathological significance. Any small hemorrhages, etc., have, if they be present, only a secondary significance.

Diagnosis.—In most cases, tetanus can be easily recognized from the peculiar convulsions and the general aspect of the disease. It might be confounded with acute meningitis, for this may cause rigidity of the neck and back; but here there are usually certain cerebral symptoms also present, such as headache and impairment of consciousness; and, on the other hand, in tetanus, trismus is an almost constant phenomenon, although exceptional in meningitis. Strychnin poisoning produces convulsions similar to those of tetanus, but they generally affect the extremities in a more marked degree. Hydrophobia is distinguished from tetanus by the ætiology, the absence of trismus, the predominance of the pharyngeal convulsions, and the greater distinctness of the individual paroxysms. The hysterical conditions similar to tetanus can usually be easily distinguished from genuine tetanus by the discovery of specific hysterical symptoms (anæsthesia, etc.).

Where trismus is the only symptom, we must guard against mistaking for tetanus the symptomatic rigidity of the jaws which occurs with severe sore throat, diseases of the teeth, or inflammation of the maxillary articulation.

Treatment.—The attempts to cure tetanus by means of an antitoxic serum (Behring, Tizzoni) are most interesting. Horses may be rendered immune to tetanus. Their blood serum then contains an antitoxin which annuls the action of the tetanus toxin as long as it circulates free in the blood. If a case of tetanus comes under treatment during the first thirty-six hours, the prospects of improvement from the injection of serum are very good. One hundred antitoxin units are injected (i. e., a vial of Behring's antitoxin) near the suspected site of infection. The injection is repeated daily for several days. If serum treatment is deferred, the chances for improvement are small, as then large quantities of tetanus toxin are already taken up by the ganglion cells, and in consequence can no longer be influenced by the antitoxin. Of course a trial of antitoxic treatment will be made whenever possible.

If the physician cannot obtain an antitoxic serum, he must look to the methods of treatment heretofore employed. We have ourselves treated all the cases we have seen in the last few years with salicylic acid (8 gr., gm. 0.5, every hour), and we believe that we have repeatedly seen a favorable influence from this remedy. In addition we like to make use of routine diaphoretic procedures, which the patients tolerate well because they usually bring relief from their painful spasms. Otherwise narcotics are to be used. Those chiefly recommended are subcutaneous injections of morphin, opium in large doses (if desirable combined with bromids), and chloral hydrate, of which 30 gr. (gm. 2) should be given two or three times a day, and the amount gradually increased. If deglutition be very difficult, the chloral may be given by enema. The above remedies diminish the irritability of the nervous centers. In curare we possess a means of lowering the excitability of the terminations of the motor nerves in the muscles. It has therefore been employed by many, but by few with success. It is difficult to say what the dose of curare is, inasmuch as the strength of different samples varies. The best way is to determine the strength of the solution to be employed by experimenting on some animal. Usually a one-per-cent solution of curare in water is employed, and an amount equal to one quarter of the contents of a Pravaz syringe is injected, the dose being gradually and cautiously increased. [Such a syringe contains about 13 minims (0.8 gr.).]

It is very desirable to put the patient by himself in a darkened and quiet chamber. Nourishment should be liquid, and lukewarm stimulants, such as alcohol and camphor, should be given from the first. Protracted warm baths may be given cautiously. We know from personal observation that such baths are very grateful to some patients.

It need hardly be said that in traumatic tetanus the primary wound should receive careful attention. [Prophylactically, suspicious wounds should be laid open and thoroughly cleansed antiseptically, and the patient given serum.] Since the tetanus bacilli remain limited in their growth to the seat of the wound, it may be indicated in the beginning of tetanus, if possible, to amputate the wounded part or to excise the wound, as in the case of a toe injury. Of course, from present experience, we cannot with certainty promise success.

CHAPTER XXI

HYDROPHOBIA

(Rabies canina. Lyssa)

Ætiology.—A peculiar infectious disease sometimes occurs in dogs, and more rarely in some other animals—viz., the wolf, fox, cat, etc. Men who are bitten by the animal may catch the disease, and thus suffer from very severe symptoms originating in the central nervous system.

Two forms of madness are distinguished in dogs—the raving madness and the quiet madness. Bollinger describes the raving form as beginning with prodromata, the melancholy stage, lasting one to three days. The animal is low-spirited, timorous, and without appetite. Then comes the stage of irritation

or of mania, in which the animal is possessed with an impulse to bite. It seems determined to run away and rove about, and it utters a peculiar howl. The dog will not touch his ordinary food, but he often swallows straw, hair, earth, bits of wood, etc. In the third or paralytic stage paralysis appears. The dog looks lean and wretched, and always dies on the tenth day at the latest. In what is called the quiet madness there is no maniacal stage. The symptoms of paralysis, affecting chiefly the hind limbs and the lower jaw, occur earlier and are sooner fatal. Marked pathological changes are not found. There are pulmonary and intestinal catarrh and passive congestion of the viscera, and the stomach often contains foreign bodies in place of the usual partially digested food.

[On the Western plains hydrophobia is said not infrequently to follow skunk bites. The bite is inflicted during sleep on persons passing the night in the open air or in tents to which the animal can gain access.]

Rabies is transferred to the human being almost invariably by the bite of some raving animal, and this animal is almost always a dog; much more rarely a wolf or cat. It is of practical importance, and it has been repeatedly confirmed, that the bite of such an animal, although still in the incubation period of hydrophobia, can convey the disease to man. The poison, which is not yet known in its pure form, is evidently contained in the saliva or slaver or in the blood of mad animals, and can, by means of these substances, be successfully inoculated in other animals. Pasteur has discovered another way to produce the disease experimentally. One takes minute portions of the brain, medulla oblongata, or some other internal viscus of a mad dog, and either injects them into the veins of a healthy animal, or trephines, and then inserts them beneath the meninges. The virulence of the rabic poison when thus manipulated undergoes, under special conditions, very peculiar alterations, which will be detailed at the close of this chapter.

About one half of those who are bitten by mad animals exhibit no subsequent symptoms. Still, this can scarcely be due to inherent immunity from the disease, and it must result chiefly from imperfect infection. Experience has shown that infection is especially apt to follow when through the bite a large quantity of the saliva of the diseased animal gets into the wound and, at the same time, small nerve branches are injured. It is believed that the poison reaches the nervous centers chiefly through the nerve channels, and to a lesser extent by way of the blood and lymphatic streams. The liability to infection is in direct ratio to the size of the bite and its proximity to the brain. Wounds of the head are therefore the most dangerous, and next to them come wounds of the hands.

The duration of incubation until rabies finally breaks out seems to vary greatly. As a rule it is about three to six months, but observers have reported instances both of shorter and of much longer duration.

Clinical History.—The disease begins with a general feeling of indisposition, anorexia, headache, and uneasiness. This last is partially explained, to be sure, by a dread of what is impending. If the bite was in the face, frequent convulsive sneezing may occur. Even now, in this prodromal stage, a marked aversion to liquids is a usual and early symptom. The attempt to swallow excites slight convulsive disturbances. Painful sensations may arise once more in the bitten place, although this has usually been cicatrized

long before; and the neighboring lymph-glands are often found to be swollen.

Only a day or two later the second hydrophobic stage begins. The especial characteristic of this consists in the peculiar attacks of tonic convulsions. The pharynx suffers most, but convulsions also attack the muscles of respiration and those of the trunk and extremities. Severe dyspnœa and a terrible feeling of anxiety and oppression accompany these attacks, so that one who has once witnessed the sight can never forget it. The convulsions always seem to be reflex, and are produced by the slightest causes, particularly by any attempt to swallow, or sometimes by the mere sight of water. They recur at gradually diminishing intervals, and last from a few minutes to half an hour. The excitement of the patient may reach the pitch of delirium or mania. The pulse is at first full and rapid, but later it is small and irregular. The temperature is usually only slightly elevated, but later it not rarely rises to 39° to 40° C. (102° to 104° F.). Shortly before death hyperpyrexia is sometimes present. There is great thirst, accompanied by burning pain in the throat. Usually there is marked salivation.

This condition lasts one to three days. Then death occurs, ushered in by violent convulsions. Death may also be preceded by a brief third stage of paralysis, during which there are no convulsive attacks. Cases of recovery in man, if they ever happen, are extremely rare.

The autopsy shows very little. The brain and medulla especially show no gross changes, so that we may conclude that hydrophobia is chiefly the effect of a toxin. The microscope has repeatedly detected very minute hemorrhages, clusters of lymph-cells around the blood vessels, etc. There have been only a few investigations of the finer microscopical changes in the motor ganglion cells of the medulla, etc.

Negri was the first to discover certain peculiar but important changes in the ganglion cells, chiefly those in the hippocampus major. They contain small round bodies with honeycomb-like structure, in whose interior there are often one or two minute vacuoles. In sections stained with eosin-methylene-blue, these Negri bodies appear red on a blue background. In all probability they are not microörganisms, but the outcome of special cell changes. They are, however, of great diagnostic significance, since they are found almost constantly and exclusively in human as well as in animal hydrophobia. The throat may present the signs of catarrh. The lungs are congested, and often œdematous. The blood is dark, with few clots. The heart, liver, and spleen are normal.

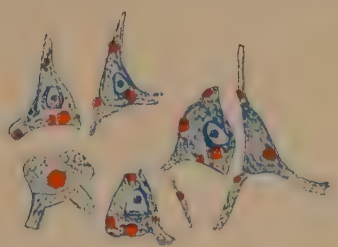


Fig. 27.—Ganglion cells from the region of the hippocampus major of a rabbit, showing Negri bodies.

Diagnosis.—The diagnosis is usually easy, particularly if we know of the possibility of infection. We are guided by the convulsions following attempts to swallow, as well as by the whole group of symptoms. Hydrophobia is distinguished from traumatic tetanus by the absence of trismus and of the characteristic tension of the muscles of the back and abdomen, by the convulsions coming in separate attacks, and by the usually greater length of incubation. Only the so-called hydrophobic tetanus (*q. v.*) bears very great resemblance

to rabies. It should be mentioned that the mere dread of hydrophobia may cause an easily excited person to have the nervous symptoms of the disease ("hysterical hydrophobia"), but of course without disastrous results. Hysteria, also, may give rise to convulsions on swallowing somewhat resembling those of hydrophobia.

Treatment.—However hopeless treatment seems, we must at least try to mitigate the patient's suffering. Narcotics accomplish this best—e. g., opium or chloral, or, most useful of all, the inhalation of chloroform. Curare has been administered repeatedly, and does seem to lessen the violence of the attacks. The attempt to find a serum-therapy for hydrophobia has unfortunately not been thus far successful.

Prophylaxis is extremely important. We cannot consider in detail the regulations (muzzling) which the government should make in order to prevent the spread of the disease. As to individual prophylaxis, every suspicious bite should be very thoroughly disinfected, and then cauterized either with carbolic acid, caustic potash, or the red-hot iron. It has also been recommended that the entire wound or scar should be excised, along with any swollen lymphatic glands which may be found in the neighborhood. Internal remedies to prevent the outbreak of the disease are probably quite useless.

On the other hand, Pasteur has recently made a series of extremely remarkable observations which have led to a special method of prophylactic inoculation against rabies in human beings. If a bit of the spinal marrow taken from a mad dog (an emulsion of the substance of the cord rubbed up in salt solution or bouillon) is introduced beneath the dura mater of a rabbit by means of trephining, the animal exhibits the symptoms of rabies after fourteen days' incubation. If in the same way a second rabbit is inoculated from the first, and so on, the virulence of the inoculated material increases gradually with every inoculation, while the period of incubation grows shorter and shorter, till it lasts but seven days. Beyond this point the period of incubation does not seem to diminish. If, on the other hand, the same series of inoculations is made on apes, the virulence of the poisonous matter does not increase, but diminishes. If dogs are inoculated with material artificially attenuated in this manner, the animals remain in good health, and furthermore acquire an immunity against more virulent inoculations, so that they may be bitten by mad dogs without becoming infected.

Pasteur has also published a still more simple and valuable method of artificial attenuation of the virus. He removes small portions of the spinal marrow of rabbits which are suffering from rabies in its most violent form produced by the above-detailed method, and these bits of marrow he exposes to air which has been wholly deprived of moisture. In this way the poison contained in the spinal marrow gradually and progressively loses its virulence, until it finally becomes inert. A portion of spinal marrow which by long drying has completely lost its original virulence is then rubbed up in sterilized bouillon and injected into some animal—for instance, a dog—and then in regular succession pieces of marrow which have been dried for shorter and shorter periods and so contain more and more of the poison, until finally the point is reached when it is possible to use for the injection pieces which are perfectly fresh and extremely poisonous, without affecting the animal's health. That is, the animal has attained immunity from the disease.

This method of Pasteur has now been employed on many thousands of human beings who were bitten by mad dogs. According to the reports, so small a number of these inoculated persons (about one per cent) have actually been attacked later by hydrophobia that we can no longer doubt the prophylactic value of Pasteur's protective inoculations.

Experiences thus far tend to show that there is no danger of exciting hydrophobia by such "protective inoculation." Ill effects from the inoculation have only been very rarely observed. There have been a few cases of acute paraplegic, motor, and sensory paralysis, but they have always terminated favorably.

At present prophylactic inoculation against hydrophobia can be practiced only in specially equipped institutions. In most civilized countries, therefore, institutions on the plan of the "Pasteur Institute" in Paris have already been established. In Germany, prophylactic treatment against rabies can be administered at any time in the Berlin Institute for Infectious Diseases and in the Hydrophobia Station connected with the Hygienic Institute at Breslau. It is of paramount importance to commence treatment as soon as possible after the bite, as immunization requires two or three weeks' treatment.

CHAPTER XXII

GLANDERS

(Farcy. Malleus)

Ætiology.—Glanders is a disease of the horse and some animals allied to it—viz., the ass and mule. It can, however, be transferred to man. It is characterized by peculiar new growths, either like nodes ("farcy-buds"), or more rarely diffuse. These are very prone to suppurate and break down. Such nodes, and the ulcers which they leave behind them, occur most frequently in the mucous membrane of the nose. In horses the purulent nasal discharge is one of the earliest and most important symptoms of the disease. Similar nodes are found in the larynx, lungs, liver, spleen, and kidneys, and often also in the skin. The cutaneous swellings and deep, crater-like ulcers belong to that form of the disease which is called "farcy." The corresponding lymphatic vessels and glands are usually much swollen. The animal has fever, grows weaker and weaker, and almost invariably dies at the end of one to three weeks.

Glanders in man is always referable to infection from a diseased animal, although in certain instances it is impossible to demonstrate the source. The disease is therefore commonest among persons who have much to do with horses —e. g., hostlers, coachmen, farmers, and cavalrymen. The virus is usually conveyed by the pus and nasal secretions of the diseased animals. A little of this falls upon some excoriation on the hand or some crack in the skin, and is absorbed. Man does not seem very liable to the disease; it is of rare occurrence.

Löffler and Schütz have discovered the specific disease-producing agent. These investigators were able to demonstrate in all the products of glanders

delicate bacilli about the size of the bacilli of tuberculosis. These bacilli can be reared artificially, and, if inoculated upon horses and other animals, they give rise to a typical attack of glanders in every instance. The bacilli of glanders have so far never been detected in the blood. It is also a very interesting observation that they rapidly lose their virulence in purified cultures outside of the living body. This is one more proof of the fact, which is lately coming more and more into prominence, that the external influences surrounding the life of bacteria greatly modify their biological peculiarities. By repeated inoculations of glanders in the horse the virulence of the bacilli rapidly diminishes, while, on the other hand, by repeated inoculations in the weasel the virulence is said to be very considerably increased.

Clinical History.—The period of incubation lasts about three to five days, and sometimes longer. The first symptoms are local, if the infection has resulted from a visible injury. There is considerable swelling and pain in this spot, and usually considerable lymphangitis in its neighborhood. In other cases, however, the disease begins with indefinite constitutional symptoms, such as fever, headache, and pain in the limbs, so that there may be some resemblance to beginning typhoid fever. The local and general disturbances increase, and the disease soon attacks other parts of the body. In the skin we see small macules or papules, singly or in groups, which soon change to pustules like those of smallpox, or to larger abscesses. These abscesses burst and discharge offensive pus, leaving behind them irregular, deep ulcers. Not infrequently the joints are swollen. The mucous membranes are also attacked; chief among these troubles are ulcers in the nose. The nose swells as if with erysipelas, and there is a purulent, foul-smelling discharge. The nose rarely escapes. The conjunctivæ, throat, mucous membrane of the mouth, and the larynx also undergo inflammation and ulceration. A violent, diffuse bronchitis develops. Sometimes there is considerable disturbance of the stomach and intestines, giving rise to vomiting and diarrhea. At the same time the constitutional symptoms become more and more severe. The patient grows stupid or delirious. In some few cases the severe cerebral symptoms are due to a purulent meningitis, perhaps through extension of the inflammation by contiguity from the nose. The fever is high, and sometimes it is quite continuous. More rarely there are chills and great elevations of temperature, as in the fever of pyæmia. The pulse is rapid and small. The spleen is seldom much enlarged. The urine may contain a trace of albumen.

In these severe acute cases the termination is almost always fatal. Death occurs at the end of two to four weeks. There are cases with a more chronic course, with tedious persistence of the troubles in the skin and mucous membranes, and milder febrile and constitutional symptoms. Such attacks appear at first tolerably favorable, but they may end fatally with persistent fever and increasing physical weakness, or they may run on for months, and at last end in complete recovery.

The *autopsy* reveals a condition greatly resembling that in pyæmia. We find abscesses in many parts, particularly the muscles and the lungs, and, next in frequency to them, the spleen, brain, and other viscera. In the mucous membrane of the nasal cavities, the pharynx and the larynx, are found nodes and ulcers such as occur in the horse. As in septicæmia, there are often numerous hemorrhages into the serous and mucous membranes. It has already

been mentioned that the specific bacilli of glanders are present in the abnormal secretions.

Diagnosis.—Without the aid of ætiological factors the diagnosis of glanders is often very difficult. Indeed, until recently there have been instances where even the autopsy did not suffice to exclude pyæmia; but now that the specific bacilli have been discovered we can clear up all doubts. We cannot here enter into a discussion of the details of bacteriological examination (inoculation of guinea pigs, preparation of cultures, agglutination, etc.). At the bedside, also, ætiology is all-important in diagnosis—e. g., exposure to infection, or occupation. The most characteristic symptoms are the nasal and cutaneous. In a case that takes a chronic course there is a possibility of mistaking the cutaneous ulcers for syphilitic or tuberculous sores.

Kalning has discovered a very interesting fact, which, for obvious reasons, has rarely been applied to man. A substance can be obtained from cultures of glanders bacilli, the so-called mallein. If this be injected in small amounts in horses sick with glanders it causes high fever, while the injection has no effect in any other animal. Mallein, therefore, has the same significance in the diagnosis of glanders as tuberculin has in the diagnosis of tuberculosis.

Treatment.—We have already implied that the treatment of acute cases is almost hopeless. We must do all we can in the way of cleanliness and disinfection to improve the local condition of the skin, the nose, and the throat. All abscesses and nodules must be opened as soon as possible and curetted and disinfected. Appropriate agents are carbolic acid and peroxid of hydrogen. Further treatment should be in accordance with the general rules for the care of severe acute infectious diseases. An inunction treatment with unguentum cinereum (gr. xxx to xlv [gm. 2 to 3] daily) has a very favorable effect. Iodid of potassium, arsenic, etc., have also been recommended. Chronic glanders swellings of the nose are at times favorably influenced by Röntgen rays, Finsen light, and the like.

CHAPTER XXIII

MALIGNANT PUSTULE

(*Anthrax. Charbon. Splenic Fever. Mycosis intestinalis. Carbunculus contagiosus*)

Ætiology.—The cause of malignant pustule is the infection of the body with a specific kind of bacilli, the *Bacillus anthracis*. This organism was discovered by Pollender in 1849, and a few years later, independently, by Brauell.

These bacilli are very minute cylinders, about as long as the diameter of a red blood corpuscle. They are found in enormous numbers in the blood and organs of animals which die of anthrax. Anilin-staining makes them more easily visible. By means of blood containing the bacilli, Davaine (1863) and others have inoculated many animals with the disease, including mice, rats, guinea pigs, cows, sheep, goats, and birds. The bacilli can also be isolated and cultivated, and then produce infection. This is proof positive that they

are the actual carriers of contagion. The rapid increase of the anthrax bacilli in the blood goes on by subdivision. In the artificial cultivations, however, the bacilli grow, as Koch has shown, into quite long threads, in which shortly appear minute, brilliant egg-shaped bodies (*cf.* Figs. 28 and 29). The threads become disintegrated, setting free the little shining ovoids, the spores of anthrax, to grow into bacilli. The bacilli can live only a relatively brief time; but the spores have unusual tenacity of existence. They may remain dried up for years, and then be brought to further development if placed in favorable conditions of heat and moisture. If the spores are transferred to animals, they develop into bacilli, and there is scarcely room to doubt that men and animals are quite as often infected by spores as by full-grown bacilli. There are facts which render it not improbable that the anthrax bacilli exist in other places than the bodies of men or animals, and may there complete their circle of development. Such places are marshes, the banks of streams, and the like. If it is possible for them to be carried by high water to the pasture lands, we have an explanation of those sudden endemic appearances of anthrax which sometimes occur in places previously free from the disease.

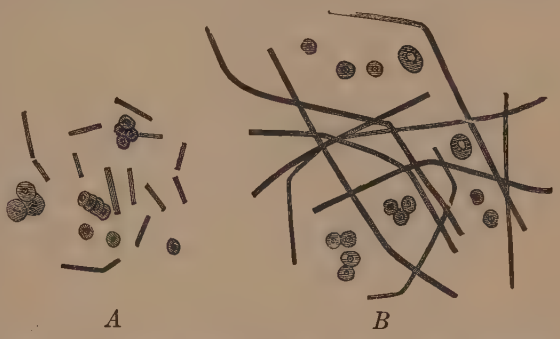


FIG. 28.—Anthrax bacilli. (From KOCH.) 650 diameters. *A*, from the blood of a guinea pig. *B*, from the spleen of a mouse after three hours' culture in the aqueous humor.

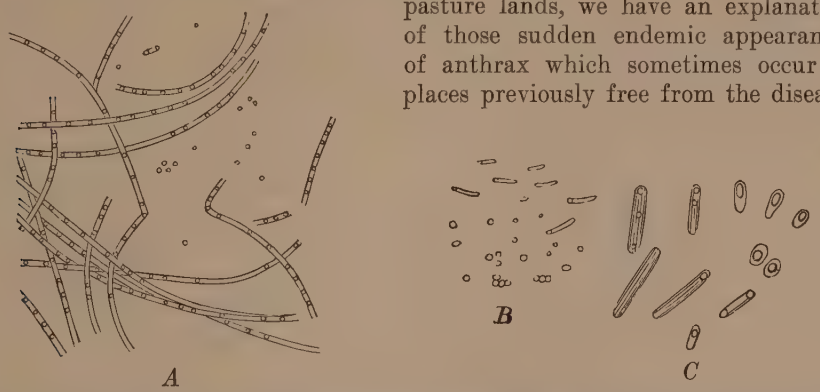


FIG. 29.—Anthrax bacilli; spore formation and spore germination. (From KOCH.) *A*, from the spleen of a mouse after twenty-four hours' culture in the aqueous humor, spores arranged like beads in the filaments. 650 diameters. *B*, germination of the spores. 650 diameters. *C*, the same, with a higher power. 1,650 diameters.

Anthrax in animals is of great practical importance because its favorite victims are the herbivorous domestic animals—viz., the cow, sheep, and horse. Among these it is terribly destructive. It is remarkable that the carnivora enjoy almost complete immunity. The disease usually runs a very acute course in animals. Indeed, it often seems like apoplexy; the apparently healthy animal suddenly falls, suffers for a few minutes from convulsions and

dyspnœa, and dies. Other cases have a somewhat longer and more intermittent course, but in these also recovery is very rare.

Human beings are probably infected in most cases by direct inoculation. Shepherds, farmers, butchers, and others who come in contact with animals suffering from anthrax, are liable to infection through any little wound or scratch upon the hands. Very often the disease is caught from hides, hair, or other parts of dead animals. In workshops and factories where wool and hides have been used which came from diseased animals, anthrax has repeatedly occurred. Curriers, rope makers, paper makers, hatters, furriers, brush workers, and those who handle horsehair and wool, are all exposed. Anthrax has also acquired the name of "rag-pickers' disease." Another way of infection, supposed to happen among animals as well as men, is through the sting of insects—e. g., flies—bringing the poison from diseased animals. It is not likely that the virus can be absorbed through the unbroken skin. It is certain, however, that the intestine may sometimes afford ingress to the infectious matter. Koch has proved this by putting spores in the food of sheep. Intestinal mycosis in man (*vide infra*) may very possibly be due to a similar mode of infection. Many cases of poisoning from eating meat have been referred to the ingestion of the flesh of animals who died from anthrax. Some observations on pulmonary anthrax seem to favor the idea that the poison may be inhaled with dust and develop primarily in the lungs.

Clinical History.—Anthrax in man has two distinct forms. These may appear in combination. The first begins with a local disorder of the skin at the point of infection—viz., the malignant pustule, or anthrax carbuncle. The second and rarer form presents the symptoms of a severe acute constitutional infection. An accompanying cutaneous disorder or other local disease is sometimes observed.

1. *Malignant Pustule.*—The malignant pustule usually comes on the hand, the arm, or the throat, and appears from three to seven days after infection. A small vesicle forms at the infected spot, grows rapidly, becomes excoriated, and usually takes on a characteristic appearance, being of a dark-bluish or black color. The surrounding parts become diffusely swollen and red. Secondary vesicles may surround the original one. In severe cases the swelling becomes more and more extensive (so-called anthrax erysipelas). Inflamed lymph-vessels or veins radiate in red lines from the pustule, and the neighboring glands are also affected. These appearances are accompanied by fever, and more or less prostration. In a favorable case the swelling subsides, the scab falls off, and there is at last complete recovery. But in other cases the constitutional infection becomes more and more prominent, and eclipses the local disorder. The fever and prostration increase. Severe intestinal symptoms appear, or else stupor, delirium, and other nervous disturbances; and death may ensue after a few days' illness.

The so-called *anthrax œdema* (malignant œdema, *œdeme charbonneux, charbon blanc*) is a form of primary anthrax of the skin differing from malignant pustule. It is seen chiefly in the eyelids, lips, and mucous membrane of the mouth and tongue, and also in other parts of the skin. In this form we see a circumscribed, doughy, œdematous swelling, in which small bullæ with sero-sanguineous contents often develop. These bullæ may become gangrenous. On the neck and trunk the anthrax œdema may sometimes be of con-

siderable extent. The other symptoms are similar to those of malignant pustule. We cannot make an absolute separation of the two forms.

2. *Intestinal Anthrax* (*"Intestinal Mycosis"*).—A quite different picture is presented by this second form, which is called intestinal anthrax (formerly intestinal mycosis) from the marked intestinal lesions. In this the cutaneous disorder, if it exists at all, is insignificant compared with the severe constitutional disturbance. It is only within a few years that the labors of Buhl, Waldeyer, E. Wagner, Leube, and others have shown that attacks of this kind have any connection with anthrax.

In cases of this sort the attack is usually rather sudden, beginning with chilliness, vomiting, headache, and languor. The diagnosis is usually very obscure at first, unless the calling of the patient suggests the possibility of anthrax. On careful examination, we may find some places where the skin is broken, or possibly a small characteristic pustule. In a case which came under our own observation a pustule had existed on the back of the right hand for some weeks before severe symptoms appeared, but had not attracted the attention of the patient at all. In this case, therefore, the constitutional infection seems to have come from the local disease. But in other cases cutaneous lesions, in the form of small carbuncles, may occur secondarily in the course of the disease. Hemorrhages into the skin and mucous membranes (especially on the gums) also occur.

Of the other symptoms, the gastro-intestinal deserve to be mentioned first. Vomiting occurs frequently, and also a moderate, painless, and sometimes bloody diarrhea. There is usually severe dyspnœa, and a marked sense of oppression in the thorax, but without objective pulmonary signs. Very soon there is collapse; the nose and extremities grow cool; the pulse is rapid, but small; and there is lividity. In a few instances tetanic or epileptiform convulsions have been observed. Œdematous swelling of the eyelids is sometimes seen. The temperature is seldom much elevated. It may be subnormal. In a few days the prostration becomes complete, and death ensues. Sometimes these severe general symptoms are associated with the signs of a circumscribed pneumonia (*vide infra*).

Milder forms apparently occur, but here the diagnosis may not be absolutely certain. We have seen a few such cases originating in a ropewalk where Russian hair was used. The constitutional symptoms were only moderately severe, the fever was mild, and recovery occurred after two or three weeks.

3. *Pulmonary Anthrax.*—Pulmonary anthrax probably arises, as we have said, from the inhalation of dust containing anthrax spores. The disease runs its course as a broncho-pneumonia, usually bilateral and associated with high fever, pleurisy, severe dyspnœa, cardiac weakness, and great general prostration. The anthrax bacilli have been found in the sputum and the pleuritic exudation, and sometimes in the blood. Most cases of this type terminate fatally in a few days.

Pathology.—In the fatal cases of anthrax the intestinal lesions are the most characteristic. Besides the signs of catarrhal inflammation, we find peculiar lesions in the mucous membrane of the small intestine, and sometimes in the upper portion of the colon. These consist of dark, infiltrated spots, with hemorrhages, the spots being somewhat larger than a silver dime. The microscope reveals numerous collections of anthrax bacilli, situated

chiefly in the lumen of the blood vessels. The spleen is usually only moderately enlarged, but dark and congested. There may be ecchymoses in the kidneys, the brain, and the serous membranes. Often there is swelling of the lymph-glands. In one case which we saw, with slight intestinal lesions, the mesenteric glands were considerably enlarged, and the bronchial lymph-glands were perfectly enormous. The bacilli are found in all the organs mentioned.

Diagnosis.—The diagnosis of malignant pustule is seldom difficult, particularly if attention is directed to the ætiology. All doubt is over if we find the bacilli. The cases of intestinal and pulmonary anthrax may be more obscure. The chief point is that the attention be directed to the possibility of anthrax by the patient's occupation, the severe general symptoms, and any pustule on the skin. For a positive diagnosis we need, of course, to find the bacilli in any pustule on the skin or in the blood.

Treatment.—1. *Prophylactic inoculation.* Toussaint and Pasteur were the first to show that the virulence of anthrax bacteria can be artificially diminished by certain external influences. If the bacilli are kept under cultivation for several weeks at an unchanging temperature of 106° to 107.5° F. (42° to 43° C.), they preserve their external appearance completely, as well as their ability to grow, but gradually lose their power of infection. Inoculations made with this "vaccine virus" produce at most an insignificant disturbance. Pasteur's discovery that the animals thus vaccinated are protected thereafter from infection with actual anthrax was most valuable. Pasteur therefore proposed that the prophylactic inoculation of sheep and other animals liable to anthrax should be undertaken on a large scale, promising the farmers that very great benefit would result. So far as experience has thus far gone, the mortality of anthrax in sheep and cattle seems actually to have been considerably diminished by the many protective inoculations which have been performed in France and Hungary.

French investigators have lately made known new methods of producing an artificial diminution of the growth and virulence of anthrax bacilli. Chauveau has found that cultures of anthrax bacilli exposed for several days to an atmospheric pressure of three to twelve atmospheres, or to compressed oxygen, lose a portion of their virulence; and that animals inoculated with bacilli thus attenuated gain an immunity to inoculations with the original anthrax poison. The statements of Arloing are very remarkable. He says that the direct play of sunlight, or even of a concentrated artificial light, upon the cultures exercises a restraining influence upon the growth and poisonous properties of the bacilli, and that inoculation material weakened in this way may be employed to render animals immune. According to the most recent researches, the serum of animals, artificially immunized, also possesses decided protective powers. These various prophylactic measures have as yet been employed on human beings only in isolated instances.

2. The treatment of malignant pustule is *surgical*. Cauterization with caustic potash, nitric acid, or carbolic acid has been found ineffective and even injurious. In mild cases rest, moist applications of aluminum subacetate, ice bags, and the like are sufficient. In severe cases experienced surgeons advocate the division of the pustule, application of the thermocautery to the circumference of its base, and the injection of tincture of iodin, in drops, into the border line between the inflamed and the healthy skin.

3. The treatment of intestinal and pulmonary anthrax must be purely *symptomatic.* In intestinal anthrax the use of calomel (gr. ij to iij [gm. 0.1 to 0.2] several times a day) is most to be recommended. Quinin (gr. vij [gm. 0.5] four times a day) and carbolic acid (gr. xv [gm. 1] a day in pills or subcutaneously) seem also to have a favorable influence on the general infection.

CHAPTER XXIV

TRICHINOSIS

(Trichinatous Disease)

The Natural History of Trichinæ.—The *Trichina spiralis,* one of the class of roundworms or nematoda, has long been known to occur occasionally in the muscles of men and certain animals; but it was not until 1860 that Zenker showed that trichinæ are capable of exciting in man a dangerous and sometimes fatal disease. Since then numerous individual cases and quite extensive epidemics have been reported; and the labors of Virchow, Leuckart, and others have taught us the anatomy and mode of development of this peculiar parasite.

The trichina appears in two shapes—as intestinal trichina and as muscular trichina. The intestinal form is a small white worm visible to the naked eye. The female is $\frac{1}{8}$ to $\frac{1}{6}$ of an inch (3 to 4 mm.) long, the male only $\frac{1}{25}$ to $\frac{1}{14}$ of an inch (1 to 1.5 mm.). They have well-developed digestive and sexual organs. The male is distinguished by two little processes at the tail. The muscular trichina (*vide* Fig. 30) is a small worm, $\frac{1}{36}$ to $\frac{1}{25}$ of an inch (0.7 to 1 mm.) long. It is found coiled up among the muscular fibers, inside a connective-tissue capsule which is often calcified.

The events in the life of the trichina are remarkable. If living muscular trichinæ reach the human stomach through the eating of trichinous pork, the capsules are dissolved, and the trichinæ, thus set free, grow in two or three days into sexually perfect intestinal trichinæ. In the uterus of the impregnated female the eggs develop into embryos, which are born already hatched. The birth of the embryos begins seven days after the ingestion of the muscular trichinæ, and seems to continue for some time. A single female is said to produce more than one thousand embryos. These latter begin their travels soon after birth, and reach the voluntary muscles. As to the routes they choose we are still somewhat in doubt. Some authorities state that the trichinæ penetrate

Fig. 30.—(From Heller.) An isolated primitive bundle with two free trichinæ in the sheath of the sarcolemma. Much enlarged.

through the walls of the intestine and the abdominal cavity into the connective tissue. Others affirm that they enter the lymphatic vessels, or exceptionally the blood vessels. Recent experimental investigations (Stäubli) certainly argue for the dissemination by the blood stream, as it was possible to demonstrate numerous embryos of trichinæ in the blood. They penetrate into the primitive fibers of the muscles, and cause them to disintegrate. Finally, they coil themselves up, attain the size of muscular trichinæ in about fourteen days, and become encapsulated. Each capsule usually contains but one, although it may inclose as many as four. The capsule is formed partly by an excretion from the trichina, and partly from the reflex hyperplasia of the surrounding connective tissue. The process of development is now complete. The muscular trichinæ seem, unlike the intestinal form, to have a very long lease of life, and usually endure till the death of their host. They are often found accidentally at autopsies. They are most abundant in the diaphragm, the intercostal muscles, the muscles of the larynx and throat, and the biceps.

Ætiology of Trichinosis.—The only cause yet known for trichinosis in man is the ingestion of trichinous raw or underdone pork—e. g., smoked ham. Swine are preëminently subject to trichinæ. They probably become infected in various ways—e. g., from the feces of human beings and swine suffering from trichinosis, or through the ingestion of the trichinous flesh of other swine. The waste of slaughter houses is often fed out to swine, and the disease thus disseminated. Many affirm that swine are also infected by eating rats infested with trichinæ, but the contrary condition, whereby rats become trichinous from eating the flesh of diseased hogs, seems more in accordance with the facts.

Clinical History.—The symptoms in man correspond in general to the developmental and vital processes of the trichinæ, as above depicted. In individual cases, however, the separate stages are quite often obscured, probably because all the parasites do not develop simultaneously, or because there are relapses. The first symptoms are gastrointestinal. At the commencement there is a feeling of pressure in the epigastrium, with nausea and vomiting. Later, diarrhea is prominent, becoming in some cases so violent as to remind one of cholera. It is not impossible, although rare, to find intestinal trichinæ in the stools. Sometimes there is constipation instead of diarrhea. In some cases the initial gastrointestinal symptoms are but slight. Frequently, even in the beginning of the disease, there is complaint of pain and stiffness in the muscles, too early for it to be due to the migration of the trichinæ.

The genuine severe muscular symptoms, due to the myositis produced by the trichinæ in the muscles, do not begin till the second week, or even later. In many cases, where the invading parasites seem to be relatively few in number, the muscular symptoms are slight, or wholly absent. In the more severe cases, however, they may be extremely violent and distressing. The muscles become swollen, firm, and hard, very tender on pressure, and very painful. The patient avoids all movements and contraction of the muscles as much as possible, lying motionless in bed, with flexed arms and with legs either extended or likewise flexed. The patellar reflex almost always disappears, and on testing the electrical reactions there is found a considerable diminution of muscular excitability to both the galvanic and faradic currents,

sometimes associated with delayed contractions, and abnormally long duration of the same after the stimulus ceases (Eisenlohr). The masseters and the pharyngeal and laryngeal muscles are attacked, so that there is difficulty in mastication and deglutition, and hoarseness. The involvement of the ocular muscles causes pain in the eyes. The condition of the diaphragm, intercostals, and abdominal muscles causes serious difficulty in respiration. There is distressing dyspnœa, and expectoration is so hampered that secretions accumulate in the air-passages. In some fatal cases of trichinosis death is due principally to this impairment of respiration. The condition may be aggravated by diffuse bronchitis or lobular pneumonia.

Third in the list of important symptoms comes œdema. It appears toward the end of the first week in the eyelids. Somewhat later it involves the upper and lower extremities. What produces it is not quite clear. It has been regarded as in part inflammatory and in part the result of occlusion and thrombosis of the smaller lymphatics. Cutaneous eruptions also develop—e. g., vesicles, wheals, petechiæ, and pustules. Frequently there is profuse perspiration, consequent upon which abundant crops of miliaria or sudamina may appear.

In well-marked cases there may be quite high fever and other severe constitutional symptoms in addition to the local disturbances already discussed. The temperature may for a time reach 104° to 106° F. (40° to 41° C.); but the fever is seldom continuous for any length of time, being usually interrupted by frequent and considerable intermissions. There are also a rapid pulse, headache, stupor, and other symptoms suggesting typhus or typhoid fever. In fact, the first case in which trichinosis was recognized at the autopsy (by Zenker of Dresden) had been regarded before death as typhoid. The urine may be albuminous, and, in rare instances, nephritis is seen. The changes in the blood are very interesting. At the height of the disease we find a marked leucocytosis, and, what is especially characteristic, a pronounced increase of the eosinophile cells in the blood. In cases with unfavorable termination the eosinophiles disappear from the blood shortly before death.

The duration of the disease varies widely. There are mild cases often unrecognized, which get well after slight symptoms have lasted two or three weeks. More pronounced cases occupy six to eight weeks, or even a much longer time. Of the more severe cases about one third prove fatal, usually from the fourth to the sixth week. Sometimes death is caused by the severity of the constitutional disturbance, but usually from disabled respiration. Even if the case ends favorably, recovery is often very tedious.

Pathology.—The autopsy reveals little that is characteristic excepting the changes in the muscles. There are sometimes the signs of hemorrhagic catarrhal inflammation of the small intestine. The spleen is not enlarged. Very often the liver is decidedly fatty. What should cause this in trichinosis has not yet been determined. The lungs often present islets of lobular pneumonia, or sometimes even of gangrene. The trichinæ are found in the muscles, beginning with the fifth week. They can be recognized by the naked eye as little whitish lines. We have already named the muscles chiefly infested. Under the microscope we see the fibers in which the trichinæ lie transformed into a fine granular mass. The nuclei of the muscular fibrillæ are greatly increased in number in the neighborhood of the coiled-up parasite. Finally,

the sarcolemma collapses, and becomes greatly thickened upon its external surface by a hyperplasia of connective tissue. The muscles also present many other degenerative changes, such as a flaky disintegration, waxy degeneration, and the formation of vacuoles. There is furthermore a marked increase of nuclei in the interstitial tissue of the muscles. Within the intestines are sometimes to be found, even after several weeks' illness, numerous living intestinal trichinæ—a fact of importance from a therapeutic point of view.

Diagnosis.—The diagnosis of trichinosis is generally not difficult, since the peculiar symptoms of the disease, especially the extensive, painful inflammation of the muscles and the œdema, occur in this way in only one other rare disease, primary acute polymyositis (*vide* Vol. II). Trichinosis is distinguished from this partly by the peculiar ætiology (affecting several people, the use of raw pork, etc.), and partly by the initial gastrointestinal symptoms. Trichinosis may also be confounded with multiple neuritis and perhaps with acute articular rheumatism, but careful observation of the patient will usually make the diagnosis clear. An absolutely certain diagnosis may be arrived at by finding intestinal trichinæ in the dejections. The blood findings, too—leucocytosis, and, above all, eosinophilia, perhaps also the demonstration of the embryos—are of diagnostic importance.

Treatment.—As trichinæ may still be alive in pork that has been smoked, or salted, or half-cooked (e. g., some sausages and meat balls), the only possible prophylaxis, therefore, as far as the individual is concerned, is to avoid all such food. A real protection for the public against the disease is also afforded by governmental microscopic inspection of meat, as already established in many places.

When an individual has become infected with trichinæ, if it is possible that intestinal trichinæ still are present, the treatment must always begin with the exhibition of purgatives, such as compound infusion of senna, calomel, or castor oil. Since trichinæ may be found in the intestines as long as eight weeks after the beginning of the first symptoms, we should not neglect local action on the intestinal contents even in the later stages of the disease. Of the remedies which are calculated to destroy the intestinal trichinæ, glycerin, which was first recommended by Fiedler, seems to be the most efficient. It must be given in rather large doses, say a tablespoonful every hour. Other drugs are much less reliable, but we will name among them benzine in the total daily dose of 1 to 2 drachms (gm. 4 to 8) in capsules, and picric acid in pills—the daily dose being 5 to 8 gr. (gm. 0.3 to 0.5).

Treatment is unfortunately almost wholly powerless against the myositic symptoms of trichinosis and their sequelæ. The muscular pains can be alleviated by narcotics, particularly morphin subcutaneously, poultices, and chloroform oil as an embrocation.[1] Protracted warm baths are excellent. Antipyrin and salicylic acid are also said to do good in many cases.

[1 Generally one part of chloroform to ten of olive oil. It is not officinal in Germany, but is weaker than the linimentum chloroformi (U. S. P.).—Trans.]

CHAPTER XXV

THE "FOURTH DISEASE"

[In 1900 Dukes described an eruptive disease, said to resemble both rötheln and mild scarlet fever and yet to afford no immunity to either. After an incubation of nine to twenty-one days the body is covered, in the course of a few hours, with a diffuse rash of a bright-red color. There are usually no prodromes. The temperature seldom exceeds 101° F. There is some glandular enlargement. Desquamation may be slight or considerable. There are no sequelæ.

The disease is generally viewed with skepticism, but at any rate Dukes's claim may be taken as an illustration of the general fact that the exanthems are not always typical.]

CHAPTER XXVI

MALTA FEVER

(Mediterranean Fever. Rock Fever. Undulant Fever)

[Malta fever is a disease of long duration characterized clinically by continued fever, profuse perspiration, constipation, frequent relapses, rheumatic or neuralgic pain, swelling of joints, and orchitis; bacteriologically, by the presence in the blood and organs of the *Micrococcus melitensis* (Bruce), and anatomically by congestion of the spleen and other organs.

It occurs around the Mediterranean, and probably on the Red Sea, the banks of the Danube, the southern Atlantic coast of the United States, and the islands of the Gulf of Mexico. Musser and Sailer have reported a case coming from the West Indies, and H. H. Smith one in Boston, in a Sicilian immigrant.

It is due to infection with the *Micrococcus melitensis,* above mentioned, which usually invades the body by being swallowed; but as mosquitoes have been found to contain the organism, they may possibly transmit it. The germ has been found in the blood, urine, and feces of the patient and in food, more particularly milk. Out of 101 blood cultures in persons suffering from this disease, 57 were positive.

On post-mortem examination the spleen is always enlarged, averaging twenty ounces. The mesenteric glands are swollen, but less so than in typhoid.

The period of incubation is regarded as six to ten days. The onset may be gradual or sudden. There is generally dyspepsia, languor, headache, chilliness, and great weakness, often accompanied with muscular pains. The fever is characterized by extreme irregularity with a great tendency to relapses. Its symptoms (says Bruce) are those commonly met with in other fevers, such as typhoid. Before the disease had been differentiated, the fever was described as resembling severe remittent malarial fever; this statement may give some

idea of the behavior of the temperature. As Notter says, it is impossible to present any one case or single chart as characteristic of the disease.

Hughes's description, quoted by Osler, is as follows: "Clinically the fever has a peculiarly irregular temperature curve, consisting of intermittent waves or undulations of pyrexia of a distinctly remittent character. These pyrexial waves or undulations last, as a rule, from one to three weeks, with an apyrexial interval lasting for two or more days. In rare cases the remissions may become so marked as to give an almost intermittent character to the febrile curve, clearly distinguishable, however, from the paroxysms of paludic infection. This pyrexial condition is usually much prolonged, having an uncertain duration, lasting for even six months or more. Unlike paludism, its course is not markedly affected by the administration of quinin. Its course is often irregular and even erratic in nature."

If the patient survives the first two or three weeks, he usually recovers. As a rule, death occurs, if at all, in the first or second week, preceded by continued high fever with a tendency to hyperpyrexia, delirium, dry tongue, and diarrhea. Very rarely death occurs at a later stage as a result of increasing debility. Mild cases may be practically well in twenty or thirty days. The average illness occupies three months, and a protracted case may extend over three years. In half the cases there are pain and swelling in the joints. Neuritis and orchitis are fairly frequent. The spleen is enlarged and tender.

The mortality is only two per cent.

As to *diagnosis,* we may obtain the specific organism by a blood culture. There is also a specific agglutination reaction. The disease is not influenced by quinin, as is malaria. It differs from typhoid in its longer duration, in the absence of rose spots, in the arthritic and neuralgic symptoms, in constipation being the rule, and in the lower mortality.

No specific mode of treatment has been as yet established. Especial attention must be paid to the diet and to bathing.]

CHAPTER XXVII

ROCKY-MOUNTAIN SPOTTED FEVER

(Tick Fever)

[Rocky-Mountain spotted fever is an infectious disease seen in the high mountain valleys of Montana, Idaho, Wyoming, Utah, Oregon, Colorado, and Nevada. It has an abrupt onset, with chill, continued fever, headache, severe pains in joints and bones, and an eruption, at first hyperæmic, later petechial, appearing on the ankles, wrists, and forehead, and spreading over the whole body.

It occurs in the spring, and attacks all ages and both sexes, but is most common in active men, because they are most exposed to infection. It is not contagious. The disease is conveyed by the bite of wood ticks, *Dermacentor venustus* and *D. modestus.* The survivor of an attack is therafter immune. There is a striking and constant difference in mortality in different localities.

In western Montana the average death rate is sixty-five to about ninety per cent, while in Idaho it rarely rises above five per cent.

Exactly how the bite of the tick imparts the infection is not settled. It is pretty certain that the disease is not a piroplasmosis. Ricketts has found in both kinds of tick a bacillus which gives an agglutination reaction with immune serum, but this bacillus is present not only in virulent ticks but also in those incapable of transmitting the disease.

The period of incubation is three to ten days, during which there is increasing malaise, with pain in the bones and muscles. Usually there is a severe chill at the onset. The bowels are constipated. There is jaundice. Bleeding from the nose, mouth, stomach, and bowels may occur. The pulse is characteristically rapid. Pneumonia may develop. The spleen is large and tender. Coma usually precedes death.

The rash appears on the second to the seventh day after the onset. It is first seen on the wrists and ankles, spreading toward the trunk. It also appears on the forehead and breast, but is especially marked on the back. In severe cases there may be superficial gangrene.

Death, if it takes place, usually comes between the sixth and the twelfth days. After two weeks recovery is likely. Convalescence is slow and begins about the fourth week.

As to treatment, attempts to obtain specific sera and vaccines are being made (Ricketts), but are not yet perfected. Meanwhile therapy is symptomatic. Free purgation is usually recommended, and water should be urged upon the patient.]

II. DISEASES OF THE RESPIRATORY ORGANS

SECTION I

Diseases of the Nose[1]

CHAPTER I

CORYZA

(Snuffles. Rhinitis. Cold in the Head)

Ætiology.—The well-known symptoms of coryza depend upon a catarrhal inflammation of the nasal mucous membrane. Although this catarrh is certainly due to infectious influences, still we cannot deny that it is one of those diseases in which taking cold is a contributory cause. Daily experience teaches us how often coryza follows an evident exposure to cold, such as wetting the feet. But the nasal hyperæmia and hypersecretion and sneezing, reflexly induced by the chill, soon pass away. In order to produce a real coryza with continued sickness, a subsequent infection is necessary. For this, however, opportunity is afforded by the changes in the mucosa brought about by the chill. The sensitiveness of the nasal mucosa to reflex or direct excitation differs widely in different persons, and on this depends the varying susceptibility of the individual to coryza. The indubitable infectiousness of many coryzas is mainly proved by the contagiousness of the disease. Handkerchiefs, kissing, or even mere social intercourse, may suffice to transmit rhinitis. Frequently the infectious catarrh starts in the nasal pharynx and thence invades the nasal mucosa. The first sensations are of burning and irritation in the throat.

Coryza may also arise from the action of chemical irritants or mechanical irritants, such as dust, on the nasal mucous membrane. The iodin coryza, which occurs from the internal use of iodin, is noteworthy. In this form iodin can easily be detected in the nasal secretion. The idiosyncrasy of many people to ipecacuanha is also well known, the very smell of it setting up a coryza, and sometimes considerable asthmatic disturbances. A severe coryza is the chief symptom, too, in hay fever (*vide infra*), which is probably due to

[1] Special treatises on the pathology and therapeutics of nasal diseases are to be found in the following works: M. Schmidt, "Die Krankheiten der oberen Luftwege," Third Edition, 1903; K. Zarniko, "Die Krankheiten der Nase, ihrer Nebenhöhlen und des Nasenrachenraumes," Second Edition, 1905; Grünwald, "Atlas und Grundriss der Krankheiten der Mundhöhle, des Rachens und der Nase," 1902. O. Chiari, "Krankheiten der oberen Luftwege." M. Hajek, "Pathol. u. Therapie d. entzündl. Erkrankungen der Nebenhöhlen der Nase," 1903, and others.

the action of the pollen of certain grasses on the respiratory mucous membrane. Finally, we must bear in mind that coryza may often be only a symptom of some other disease, such as measles, syphilis, or glanders, and that severe purulent inflammation of the nasal mucous membrane may be excited by the presence of the secretion from a gonorrheal or blennorrheal conjunctivitis.

Symptoms.—The symptoms of coryza are in most of the milder cases of a local nature only. The secretion is troublesome; at first it is scanty and mucous, but later it becomes more abundant and watery; and sometimes it is purulent. The nasal passages are not infrequently closed from the swelling of the mucous membrane. The patient necessarily has to breathe through the mouth, which explains the well-known nasal speech. This closure of the nares may give rise to dangerous attacks of dyspnœa in children, especially in infants, who have to breathe through the nose when sucking at the breast. The sense of smell is always diminished. The local sensations of pain and burning are due chiefly to a mild inflammation of the skin of the nostrils and upper lip set up by the irritation of the secretion. The irritated condition of the inflamed mucous membrane occasions a feeling of tickling and itching in the nose, and frequently by a reflex action violent sneezing. The symptoms are more severe if the cavities adjacent to the nose are attacked by catarrh, and if in them accumulations of secretion occur. Marked pain in the forehead occurs in catarrh of the frontal sinuses. The sinuses of the ethmoid and sphenoid bones, and the antrum of Highmore, may also be implicated. Much more frequently a severe coryza sets up inflammation in adjacent mucous membranes. Thus we find, following coryza, conjunctivitis, affection of the ear, sore throat, or laryngitis. In persistent coryza eczema is not infrequently excited on the skin of the upper lip, and mention has already been made of the fact that coryza may sometimes act as the exciting cause of erysipelas.

In severe coryza we may sometimes have quite a marked general disturbance, and often slight elevations of temperature. The "feverish cold" of children, for instance, is well known. Brief mention may be made, also, of a peculiar form of coryza. It consists in sudden attacks of a very profuse discharge of a watery secretion from the nose. Such attacks are probably due to nervous influences. Therapeutic measures are of little avail.

Treatment.—Special treatment is usually unnecessary, for most cases recover of themselves in a few days. It seems doubtful whether the internal use of quinin in fresh coryza is of service, as is claimed. With abundant secretion, especially in fresh cases, Hager's "coryza remedy" (as an inhalation) is worthy of trial; this consists of ten parts each of alcohol and carbolic acid, and five parts of ammonia water. Painting the nasal mucous membrane with a solution of cocain (two to five per cent) is also greatly praised [but it may lead to the formation of the cocain habit]. A remedy that apparently is often effective is "forman" (forman cotton, forman pastils). In the presence of warm water, this drug splits up into formaldehyd, menthol, and hydrochloric acid. It should be inhaled through the nose. When the secretion forms abundant dry scabs, an attempt should be made to wash them out by injections of warm fluids, such as warm milk. The upper lip and the nostrils should be smeared with vaselin or simple ointment to protect the skin from the action of the secretion. Only in the rare cases of a

severe purulent catarrh is energetic local treatment of the nasal mucous membrane necessary. Here we may use douches or sprays of astringents like tannin or alum, or let the patient snuff them up, or we may apply caustics like nitrate of silver. In children who cannot blow the nose, it is advisable to cleanse the nose frequently with a small sponge and a one-per-cent solution of boric acid.

HAY FEVER (HAY ASTHMA)

The so-called hay fever (*catarrh æstivus*) is of frequent occurrence in England and North America, although rarer in Germany. It usually affects men in middle life, less often women. Some individuals are peculiarly liable to the disease. For them an attack may be produced merely by walking across a meadow or near a grain field at that season when the grasses are in bloom—i. e., about May to July. As already intimated, it is supposed that the grains of pollen excite the disease, being diffused in the air and thus drawn into the nostrils. At any rate, they have repeatedly been found in the nasal secretion and also in the tears of affected persons. The *symptoms* consist in a very severe coryza, with burning of the nose and violent sneezing. The erectile tissue of the nose is probably acutely swollen. Usually these symptoms are accompanied by a well-marked conjunctivitis with œdema of the eyelids. In severer cases there is, furthermore, a catarrh of the larynx and bronchi. There is frequently a tendency to violent attacks of asthma ("hay asthma"), especially at night (see the chapter on bronchial asthma). The *treatment* consists first in avoiding the cause by change of residence, as by going to the seashore. For the nasal catarrh, douches are most to be recommended, such as a solution of 1 part of quinin to 500 to 1,000 parts of water, or a solution of carbolic acid, etc. Some authors praise massage of the nasal mucosa and the topical use of cocain and suprarenin solutions. Of the internal remedies, sodium or potassium iodid should be tried in pronounced asthmatic conditions. A peculiar method of treatment has been proposed by Dunbar. He produces an alleged antitoxic serum by the action of pollen grains on animals, and asserts that the penciling of this serum on the nasal mucosa or the use of a snuff impregnated with it (pollantin) very materially lessens the suffering of hay-fever patients. The practical results of this method are not entirely harmonious.

CHAPTER II

CHRONIC RHINITIS

(Rhinitis chronica hypertrophica et atrophica ozæna)

1. **Chronic Hypertrophic Rhinitis.**—It is in many cases impossible to determine the causes of hypertrophic rhinitis. Sometimes the condition seems to develop as a sequel to frequently repeated nasal catarrh, although in this case the relation is often reversed—it being the chronic rhinitis which

occasions a predisposition to the frequent acute exacerbations of the catarrh. Certain diatheses (anæmia, scrofula) appear to influence the development of the disease. This is also true of occupations which expose the individual to dust or smoke, and sometimes true of malformation of the nose (for instance, deviation of the septum) and perhaps also of hereditary predisposition.

The anatomical changes consist of a slow but progressive swelling and hypertrophy of the mucous membrane. This seems spongy, and of a red or reddish-gray color. The greatest change is almost always found over the inferior turbinated bone, and next to that over the middle turbinated. In advanced cases the mucous membrane presents rough, uneven swellings, and even polypi. These changes are often visible upon inspection of the nostrils anteriorly, but they may escape discovery until a rhinoscopic examination of the posterior choanæ is made.

The disturbance occasioned by chronic hypertrophic rhinitis may be very considerable. Respiration through the nose is obstructed, the voice becomes nasal, the senses of smell and taste are impaired. The nasal secretion is for the most part increased, but it may be diminished. Often there is a tendency to nosebleed. Many patients complain also of headache.

The frequent involvement of neighboring organs is important. This applies particularly to the ear. Deafness is caused both by the obstruction of the openings of the Eustachian tubes, and not infrequently also by extension of the catarrh to the lining membrane of the tubes and the middle ear. Very frequently the disease is associated with chronic nasopharyngitis or pharyngitis. The visible portion of the nose is not infrequently affected, as shown by redness and swelling of its tip.

A fact of especial interest is that such a diseased state of the nasal mucous membrane may give rise to reflex neuroses (Voltolini, Hack, and others). Although, in our opinion, many of the specialists of the nose go too far in this direction, there is no room for doubt that attacks of migraine, vertigo, certain varieties of headache, and, above all, many forms of bronchial asthma, may bear a close relation to diseases of the nose. We shall revert to this point later on. (See especially the chapter on bronchial asthma.)

The *treatment* of chronic hypertrophic rhinitis, in order to be successful, demands complete destruction and removal of the hypertrophic portions by means of the galvano-cautery. For particulars, we must refer to the directions of specialists. Only in milder cases does benefit follow penciling with cocain, nasal douches, and the insufflation of a powder composed of gr. $\frac{1}{6}$ to $1\frac{2}{3}$ = 0.05 to 0.1 gm. nitrate of silver to 3ijss. = gm. 10.0 of common starch or the like.

2. **Simple Chronic Atrophic and Fetid Atrophic Rhinitis. Ozæna Simplex.**—The disease consists in a slow, progressive atrophy not only of the mucous membrane with its vessels and glands, but finally also of the bones, and this atrophy is not preceded by hypertrophy. Thus the nasal cavities become abnormally large. The turbinated bones grow smaller and smaller, so that finally they are represented merely by narrow ridges. Furthermore, the scanty purulent secretion has a tendency to dry up and form adherent greenish-yellow scabs and crusts, which undergo a peculiar putrefactive decomposition and give rise to an unbearable stench. We do not yet know what special form of bacterium causes this putrefactive decomposition of the secretion.

When this very characteristic and extremely repulsive stench occurs in the nose, we usually give the affection the brief name of ozæna (ὄζειν, to stink), but in the other and practically less important cases we speak of a simple atrophic rhinitis. The latter may sometimes change into true ozæna.

Ozæna generally develops in childhood. It usually begins insidiously, but in other cases apparently is a sequel of some acute disease, such as measles, etc. Anæmia and scrofula deservedly rank as important predisposing causes. Störck's view that ozæna is usually connected with syphilis in the parents is not sufficiently supported. It is worthy of note that patients with ozæna often have from birth a nose with a flat, broad bridge, which is perhaps a family trait, favoring the development of the disease.

The subjective symptoms are often not marked. This is partly explained by the fact that the patient has usually completely lost his sense of smell, but for that very reason the discomfort of his friends may be the greater. The feeling of dryness in the nose may prove annoying and there are often complaints of headache and of pressure in the eyes. Inasmuch as the nasopharynx and the posterior pharyngeal wall are almost always implicated in the process, the patient often suffers from hacking and a tendency to cough and vomit. Such portions of the secretion as are swallowed sometimes give rise to a considerable chronic disturbance of the stomach. Upon physical examination we are first struck by the unusual breadth of the nostrils. With the rhinoscope the extent of the atrophy is still better seen. The mucous membrane is pale or slightly red, and covered with dry scabs. Sometimes superficial ulcers are formed. Usually, as we have said, the superior portion of the pharyngeal mucous membrane shares in the disease. The posterior wall of the pharynx is seen to be atrophied, smooth as if it were varnished, and often covered with crusts. The process may involve the soft palate, and even the larynx, and not infrequently the disease is associated with inflammation of the middle ear.

It should be added that the true ozæna must not be confounded with other processes which likewise give rise to a foul smell from the nose. Tuberculous disease of the nasal mucous membrane and nasal bones is not rare, particularly in "scrofulous" children (Demme); nor should we forget the syphilitic affections of the nose, tertiary and hereditary syphilis.

Treatment.—An alleviation of ozæna can be accomplished only by the aid of local applications as prescribed by specialists. Even then the treatment is a prolonged one, and demands much patience on the part of both patient and physician. A complete cure of atrophic rhinitis is impossible. Besides local applications, we must also bear in mind the necessity of constitutional treatment.

The object of local treatment is to remove the secretion in order to get rid of the bad odor. Nasal douches, with disinfectant solutions, such as permanganate of potassium (1 to 3,000), or phenol, or boric acid, etc., are here most used. The solution is carefully injected into the nose (best, by means of a small rubber ball having a short rubber tip—so called "Heller's nasal syringe"), or the fluid is allowed to run gently into one nostril from an irrigator while the patient keeps his head bent forward; it then runs through the nasopharynx and out through the other nostril. The patient soon learns to retain the fluid in the pharynx and eject it from the mouth. All nasal

douches must at first be used with care and under the eye of the physician. The fluid should be injected at the lowest pressure possible, so that none of it may enter the adjacent cavities or the Eustachian tube. Furthermore, all solutions used as a douche must be lukewarm—90° to 95° F. (25° to 28° C.). Besides the regular use of douches, the insufflation of powders, such as boric acid, aceto-tartrate of aluminum, aristol, zinc sozoiodid, etc., is sometimes employed. The insertion of tampons of dry absorbent cotton is to be recommended; under their use the secretion dries less easily and the odor is diminished. These tampons should be changed daily. It is advantageous to medicate the tampons with a one-per-cent solution of creolin or with Peruvian balsam or some similar drug. Tincture of iodin is also recommended. Of late many attempts have been made to treat chronic nasal catarrh by the galvano-cautery. With regard to the details of this as well as of other methods, we must refer to special treatises on the subject.

CHAPTER III

NOSEBLEED

(Epistaxis)

Although in many cases nosebleed is only a symptom, still we are justified in a short description of it, partly because frequently repeated nosebleeds often first call our attention to some other existing disease, and partly because the treatment is of practical importance.

Many persons are subject to habitual nosebleed, which comes on either from slight causes, from violently blowing the nose, from physical exertion, from overheating, or even without any special cause. This habitual nosebleed is sometimes, but by no means always, the sign of a general hemorrhagic diathesis, which is hereditary in many instances. (See the chapter on hemophilia.) In other cases the nosebleed is the result of some chronic disease. It occurs especially in leukæmia, in disease of the heart, in contracted kidney, and as a symptom of the so-called hemorrhagic diseases, such as scurvy, purpura hemorrhagica, etc. Finally, diseases of the nose itself may give rise to hemorrhage. The periodic occurrence of nosebleed in young girls as a form of so-called "vicarious menstruation" has often been described, but we must always be very guarded in arriving at this assumption. Nosebleed sometimes occurs at the beginning of many infectious diseases, especially typhoid and scarlet fevers, etc., and in septic affections. We may add that the point of hemorrhage is very frequently at the anterior lower end of the cartilaginous septum (Kiesselbach).

In many cases nosebleed is a very transitory symptom, wholly without danger, and in one sense it may even be advantageous. Headache, or a feeling of fullness in the head, is often improved after an epistaxis. Nosebleed is dangerous, however, when it takes place in those who are already weak and anæmic, or when it is so persistent and abundant as to cause a marked general anæmia. The latter is recognized by the pallor of the face, by the appearance of general weakness, by vertigo, tinnitus, and a weakened pulse. In such cases

the physician's interference is always necessary. In every case of nosebleed it is important to examine the posterior wall of the pharynx in order to see whether the blood is not flowing backward from the posterior nares. The hemorrhage is often thought to stop when no more blood comes from the nostrils, and yet the blood keeps flowing posteriorly.

In every severe nosebleed rest is the chief thing to be enjoined, and the patient must be told to avoid unnecessarily blowing, wiping, or drying the nose. By quietly and persistently closing the nostrils with a handkerchief a thrombus is often formed without any further medication, and the bleeding stops. The application of cold water (iced water), in which a little vinegar may be put, is a good thing. An effective hemostatic is adrenalin (applied in 1-to-1,000 solution). If the bleeding does not stop, we may next try a tampon of common absorbent cotton or styptic cotton, or, still better, of strips of iodoform gauze in the nostril from which the blood comes. Penghawar Djambi, a plant fiber, appears to be a very good hemostatic. If this does not succeed, the posterior nares must be plugged by means of a "Bellocq's cannula." In case of emergency we may use an elastic catheter, which is passed through the inferior meatus into the pharynx and out by the mouth. The tampon is fastened to the catheter and brought up into the posterior nares by drawing the catheter back through the nose. Internal remedies to check the flow of blood (ergotin, hydrastis, etc.) are very uncertain in their action.

SECTION II

Diseases of the Larynx

CHAPTER I

ACUTE LARYNGEAL CATARRH

(Acute Laryngitis)

Ætiology.—Taking cold plays a prominent part in the popular ætiology of acute laryngeal catarrh. Its influence cannot be wholly denied, for it is entirely in accord with our experiences otherwise that a mucosa which has been injured by chilling is rendered thereby more receptive for infection. The disposition to laryngitis differs very much in different people, so that some take a catarrh much more easily and more frequently than others. Besides cold, direct irritants which attack the laryngeal mucous membrane often set up a laryngitis; among these are in particular the inhalation of smoke and of noxious gases and vapors. Many laryngeal catarrhs, too, arise from excessive speaking, shouting, or singing, particularly if other injurious influences act on the larynx at the same time. Finally, laryngitis may appear as a complication or as a secondary affection, in other diseases, especially in measles, less frequently in typhoid, scarlet fever, and erysipelas. Catarrh of the larynx is very often combined with catarrh of the nose, the pharynx, and the larger bronchi.

Symptomatology.—Although the symptoms of laryngitis usually make the diagnosis easy and certain, yet an accurate understanding of the extent and intensity of the catarrh can be obtained only by a laryngoscopic examination,[1] which therefore should be employed in every severe case. The laryngeal mirror shows a decided reddening and swelling of the mucous membrane, varying with the intensity of the catarrh, and most marked on the true and false vocal cords and between the arytænoid cartilages. We often see small collections of mucus here and there on the membrane. In individual cases different parts of the larynx are especially affected. In intense inflammations superficial erosions are often met with, especially on the vocal cords. In other cases the mucous membrane shows a grayish-white coloring in some places, apparently due to a thickening of the epithelium. Small hemorrhages in the mucous membrane are also occasionally seen. Very often we see on phonation an incomplete closure of the glottis, so that a little oval space is left between the vocal cords. This slight "catarrhal paresis of the vocal cords" is probably of muscular origin, and depends chiefly upon an affection of the thyro-arytænoid muscles.

Of the other symptoms of laryngeal catarrh, hoarseness is particularly to be mentioned, for in many cases the diagnosis of laryngitis may be made from this alone. It is either due directly to the anatomical changes of the cords, or to the paresis just mentioned. The degree of hoarseness is of course very different in different cases, and varies from a simple "roughening" or "deadening" of the voice to a complete loss of voice (aphonia).

The cough in laryngitis may be very severe, and is often recognizable by its harsh, hoarse ring as a "laryngeal cough." It is usually dry at first, and later on it is associated with a scanty mucopurulent expectoration, which is sometimes tinged with blood.

Pain in the larynx is generally only moderate. The subjective symptoms consist chiefly of a disagreeable feeling of itching, burning, and dryness in the throat. After prolonged speaking, however, the pain in the larynx may sometimes be quite severe. External pressure on the larynx is often somewhat painful. Difficulty in swallowing, when it occurs, is due usually to an accompanying pharyngitis, but it may also be dependent upon an affection of the epiglottis and the arytænoid cartilages.

The general health is affected in very different degrees. Many patients feel quite well except for the hoarseness, while others are affected with considerable debility, mild headache, and even at times slight febrile disturbances. Of late years we have frequently seen cases of primary acute laryngitis, with great hoarseness and marked catarrhal inflammation of the upper part of the larynx, especially of the vocal cords. These cases begin with high fever (over 104° F. [40° C.]) and quite severe general symptoms, and make a complete recovery in a week or two. They apparently have an infectious origin, and are perhaps connected with influenza.

Dyspnœa is not present in the common laryngitis of adults, even if there is

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[1] More extensive observations on laryngoscopy and on many details of the pathology of laryngeal diseases, which have been carefully investigated by specialists and which cannot be mentioned here, are to be found in the following works: Schrötter, "Vorlesungen über die Krankheiten des Kehlkopfes." P. Heymann, "Handbuch d. Laryngologie u. Rhinologie." O. Chiari, "Krankheiten des Kehlkopfes u. der Luftröhre." Grünwald, "Grundriss d. Kehlkopfkrankheiten u. Atlas d. Laryngoscopie," etc. See also the literature given on p. 164.

decided swelling of the false vocal cords or of the ary-epiglottic folds. There is, however, a severe form of acute laryngitis, the so-called *laryngitis hypoglottica acuta gravis* (*chorditis vocalis inferior*), affecting not only children, but adults, in which well-marked symptoms of suffocation may be present. In this form there is an acute, very well-marked swelling of the mucous membrane in the inferior, subchordal, laryngeal space, which leads to a stenosis. The rare cases of phlegmonous inflammation of the larynx may also cause considerable stenosis and dyspnœa.

In children, however, on account of the greater narrowness of the child's larynx, symptoms of stenosis are not rare even in the milder forms of laryngitis, and therefore they have led to the establishment of a special disease, the so-called false croup.

False Croup.—The false croup (*laryngitis stridula*) of children usually follows a slight coryza. A harsh, hollow, ringing cough comes on, almost always suddenly and usually at night, by which the child is awakened out of sleep. The paroxysms of coughing are broken by long-drawn, noisy inspirations. The child is anxious and restless, the respiration is labored. The accessory muscles of respiration are brought into action, but the deep inspiratory retraction of the lower intercostal spaces and the epigastrium shows how imperfectly the air enters the lungs. The pulse is small and rapid. The attack lasts several hours, when the cough gradually becomes looser and the breathing easier. Finally, the child falls asleep and usually wakes the next morning quite lively and playful, a slight cough being the only relic of the terrifying events of the night before. The next night the same severe attack may be repeated, and perhaps for two or three nights more. After that there remains, as a rule, nothing but a slight catarrh, which completely disappears in a week or two. The anatomical cause of false croup is acute laryngitis with marked swelling of the mucous membrane on and below the vocal cords. In the narrow space of the child's larynx this speedily causes marked stenosis, and the individual attacks are probably excited by the accumulation and drying of the secretion during the night. There is never any true croupous-diphtheritic change to be seen either in the pharynx or the larynx. It is remarkable that many children, and sometimes several children of the same family, have a specially marked predisposition to false croup. The statement, therefore, that a child has had the croup several times almost always means that it has had this form of false croup just described.

Acute laryngitis lasts only a few days in mild cases, and a week or more in severe cases. With improper care and unreasonable conduct on the patient's part an acute catarrh may run into the chronic form. We hardly ever see a fatal result in adults, even in the severe form, and in children false croup very rarely has an unfavorable termination unless the child is extremely weak or rachitic.

Treatment.—The treatment of acute laryngitis requires that especial attention be paid to the removal of all injurious influences. In every severe laryngitis the patient should stay in his room, and children are better off in bed. The patient should talk as little as possible. In all severe cases smoking, too, is to be forbidden. It is a good plan to furnish plenty of warm drink. Hot milk, mixed with Seltzer or Ems water, is readily taken by most patients. If there is a steam atomizer at our disposal, we may let the patient inhale simple

steam, or a one- or two-per-cent solution of common salt. Inhalations of astringents are usually unnecessary. The patient may also breathe steam without any special apparatus. When there is marked irritation from coughing we may give a little morphin. With more marked local symptoms, especially if there is much pain on swallowing, from swelling of the epiglottis and the mucous membrane over the arytænoid cartilages, the patient may suck pieces of ice slowly. In severe cases of acute laryngitis, with evident symptoms of stenosis, ice must be energetically used as an internal and an external application. Sometimes, too, a few leeches applied in the region of the larynx afford distinct relief. Among external applications a mustard plaster over the front of the neck is to be recommended when there are marked local symptoms. Cold, wet compresses about the neck are also of advantage in all cases.

In the false croup of children we should use, as a rule, the same treatment as has just been described. The child should take plenty of warm drink, inhale warm steam or salt solution, and a mustard paste or hot poultices should be applied to the neck. An ice bag on the neck is sometimes useful. We should be rather cautious with regard to the favorite treatment with emetics. such as ipecac and sulphate of copper, although it cannot be denied that they sometimes work very well in severe dyspnœa.

These means are entirely sufficient for the treatment of acute laryngitis. It is only exceptionally that we find ourselves led to employ in acute laryngeal catarrh an energetic local treatment of the laryngeal mucous membrane, like painting with a 1-to-15 solution of nitrate of silver.

We must bear in mind that a rational hardening process is of distinct prophylactic value in persons, especially in children, with a recognized tendency to laryngitis, sore throat, etc. The best method is to bathe the neck and chest with cold water regularly morning and night.

[A mild emetic can do no possible harm in false croup, and very often cuts the attack short. The application of a sponge, moistened with water as hot as the child will bear, to the region of the larynx deserves mention.]

CHAPTER II

CHRONIC LARYNGITIS

(Chronic Laryngeal Catarrh)

Ætiology.—Chronic laryngitis develops from an acute catarrh, or comes on gradually from the action of injurious influences on the larynx (see the preceding chapter). Chronic laryngitis, therefore, is in many cases due to the occupation, and is seen especially in singers, public speakers, criers, innkeepers, workmen exposed to dust, etc. It is very frequent in drunkards, and in such cases it is almost always associated with a chronic pharyngitis. It is probably not true that, as frequently stated, too long a uvula may set up laryngitis by irritation of the entrance to the larynx.

Symptomatology.—A laryngoscopic examination is very desirable in acute laryngeal catarrh, but it is the physician's absolute duty to make one in chronic laryngitis, for only too frequently a persistent hoarseness is referred

to a "simple" catarrh when the laryngoscope gives quite another cause for it, such as paralysis of the vocal cords or new growths. Furthermore, we must always remember that a chronic laryngitis may be a complication of tuberculosis, syphilis, or chronic nephritis. We should never omit a careful examination of the rest of the body as well as the larynx.

The laryngoscopic appearance in chronic catarrh may be so like that in an acute catarrh that we cannot distinguish between them without the history obtained from the patient. The redness of the mucous membrane, however, is usually less intense, and the vocal cords have more of a dirty grayish-red appearance. In most cases there is also considerable swelling of the mucous membrane as well as redness. Much more rarely we find atrophic conditions, resembling those of ozæna and sometimes associated with it. Quite frequently in persistent catarrhs a thickening of particular parts of the mucous membrane is developed, especially of the folds between the arytænoid cartilages. This swelling is of practical importance, because it furnishes a mechanical hindrance to the closure of the arytænoid cartilages, and in that way contributes to the development of the hoarseness. We also find marked thickening of the false vocal cords (especially in public speakers and preachers) and of the true vocal cords. Virchow described, under the name of *pachydermia laryngis*, a condition of the larynx seen especially in drinkers and characterized by a thickening of the epithelium over the whole laryngeal mucous membrane. Türck has described a peculiar form of chronic laryngitis, in which rough prominences are formed in the middle of the true vocal cords, under the name of *chorditis tuberosa*. We not infrequently find in chronic catarrh superficial erosions, especially on the vocal cords. We never see actual ulceration in simple laryngitis. We also very often see a disturbance of motion of one or both vocal cords, especially incomplete closure, due sometimes to muscular paresis and sometimes to mechanical conditions.

The other symptoms of chronic laryngitis are hoarseness, cough, and abnormal sensations in the larynx. The hoarseness is of every degree, from mere roughness, frequent "cracking" of the voice, to almost complete aphonia. The cough is ringing, hoarse, deep, and rough. The expectoration is scanty, usually simply mucous, but sometimes a little bloody. The subjective sensations in the larynx are a feeling of burning and itching, and of dryness and tickling. They usually increase after any protracted use of the voice.

We must also mention as a peculiar and very rare but practically important form of chronic laryngitis the *chorditis vocalis inferior hypertrophica* (Gerhardt), or *laryngitis hypoglottica chronica hypertrophica* (Ziemssen). In this form there is a very gradual hypertrophy, and finally a contraction of the mucous and especially the submucous connective tissue in the inferior laryngeal space. More rarely the same changes are seen in the upper part of the larynx. The special symptom of the disease, besides a chronic hoarseness, is the appearance of a gradually increasing stenosis of the larynx. The respiration is always labored, the inspiration noisy and protracted. In many cases there are at times such attacks of suffocation that life can be prolonged only by tracheotomy. The diagnosis requires the aid of the laryngoscope. We see beneath the glottis a little fissure surrounded by the thick and swollen mucous membrane of the laryngeal walls.

The nature of this affection is not yet fully explained. It often seems to

be a simple chronic hypertrophic inflammation, but in other cases Schrötter considers it the same bacillary disease of the mucous membranes, which is known as *rhinoscleroma* when it occurs in the nose.

Treatment.—The treatment of chronic laryngeal catarrh is always a tedious and laborious task, the success of which depends in great measure upon the good will and energy of the patient. In the first place, then, we should endeavor to remove as far as possible those injurious influences which have excited and kept up the catarrh. It is often easier to give good advice here than to follow it. Nevertheless, it is the duty of the physician to impress upon the patient the urgent necessity of taking care of the larynx, and to forbid as far as possible all protracted speaking, singing, staying in smoke or dust, smoking, and the use of alcohol.

Local treatment takes the second place. Among the most useful means to employ are sprays of astringent solutions, such as a one-per-cent solution of either tannin or alum. If there is much secretion of mucus, turpentine inhalations are advisable. When there is great irritability of the larynx, the patient may also inhale vaporized narcotics, a mixture of fifty parts of cherry-laurel water with a thousand parts of water, or a four-per-cent solution of bromid of potassium. The inhalations should be used two or three times a day, and last about five minutes each time. Direct applications to the larynx are much more effective than inhalations, but these can be employed only by the aid of a laryngeal mirror. Of these we use, first of all, nitrate of silver, at first in a weak solution (1 to 50); later in a more concentrated form (1 to 10, or even 1 to 5). These applications are made every two or three days. Besides nitrate of silver, the larynx may also be painted with pure tincture of iodin, or with iodin and glycerin (iodin, 4 parts, iodid of potassium, 6 parts, and glycerin, 500 parts), or with concentrated solutions of alum or tannin. (See formulæ in the Appendix.) The importance of local treatment must not be overestimated. At all events, too prolonged irritation of the laryngeal mucous membrane should be avoided.

Water cures are also often prescribed in chronic catarrh of the larynx. These are of advantage from the avoidance of ætiological factors, and from the good air. Empirically, we prescribe, especially for "full-blooded" patients, the cold sulphur springs, such as Nenndorf, Eilsen, or Weilbach, or the sulphate-of-sodium waters, such as Carlsbad and Marienbad, while we send those of delicate constitutions to Ems, Salzbrunn, Salzungen, Reichenhall, etc.

The treatment of laryngitis hypertrophica, when it leads to stenosis, must be mechanical. Schrötter, in particular, has devised several methods of dilating the stenosis gradually by the introduction of bougies and harder dilators. The details of this treatment are to be found in the newer special works.

CHAPTER III

LARYNGEAL PERICHONDRITIS

Ætiology and Pathological Anatomy.—The inflammation of the perichondrium of the laryngeal cartilages is in very rare cases apparently a primary disease. It is much more frequently secondary to other laryngeal affections,

especially tuberculosis and syphilis of the larynx. Furthermore, it develops secondarily in severe acute diseases, most frequently in typhoid fever, more rarely in smallpox, diphtheria, etc. Superficial ulcerative processes in the mucous membrane often precede the perichondritis in these cases, and the participation of the perichondrium in the inflammation arises from their gradual deepening. Anatomically, we have to do, as a rule, with a purulent inflammation, which usually leads to the formation of circumscribed abscesses. Most laryngeal abscesses have their origin in the perichondrium.[1] The perichondrium is in part destroyed by the abscess and in part elevated from the cartilage. The cartilage then becomes necrotic, breaks in pieces, and is expelled in small particles or in masses.

Perichondritis occurs most frequently in the cricoid and arytænoid cartilages, much more rarely on the internal or external surface of the thyroid cartilage. Hence we distinguish an internal and an external perichondritis. A perichondritis of the epiglottis has also been repeatedly observed.

Symptomatology.—In the rare cases of primary perichondritis, marked laryngeal symptoms are speedily developed in a person previously healthy. These symptoms are pain and tenderness on pressure over the larynx, hoarseness, and cough; and to them are usually soon added the signs of a dangerous stenosis of the larynx. In secondary cases, which occur almost always in patients who are already seriously ill, the symptoms of stenosis are often the first to point to a severe disease of the larynx. On laryngoscopic examination, besides the general redness and swelling in particular places, we can sometimes recognize a circumscribed prominence of the mucous membrane caused by the abscess. If the abscess be already broken we can see the abscess cavity and sometimes the cartilage lying free within it. In most cases we find a considerable collateral œdema of the surrounding mucous membrane, which œdema frequently has a greater share in the production of stenosis than has the primary affection itself. The dreaded œdema of the glottis (œdema of the ary-epiglottic ligament) in typhoid, tuberculosis of the larynx, etc., is usually due to perichondritis of the cricoid or arytænoid cartilages. Finally, we can see with the laryngoscope, especially in perichondritis arytænoidea, a considerable disturbance of motion of the affected arytænoid cartilage, and also of the vocal cords.

Laryngeal perichondritis terminates fatally in a great number of cases from the development of stenosis. In thyroid perichondritis the pus may sometimes gravitate, causing a severe purulent inflammation of the mediastinum. In other cases the most threatening symptoms may be averted for a time, but the primary disease, such as tuberculosis, finally comes to an unfavorable termination. In the rare cases in which recovery occurs after primary perichondritis or after the termination of the primary disease, such as typhoid, this recovery is often incomplete, since a chronic stenosis of the larynx remains from the ensuing cicatricial contractions.

Diagnosis.—The diagnosis is usually obscure during the first period of severe symptoms of stenosis, since it is difficult to make a laryngoscopic examination, and it is also not always easy to determine the condition. We

[1] True submucous abscesses, the so-called phlegmonous laryngitis, occur only in very rare cases.

are usually justified, however, in making the diagnosis, if in those diseases which we have mentioned, in which we know by experience that a perichondritis quite frequently occurs, the danger of suffocation arises in addition to the other laryngeal symptoms. It is of practical importance to recognize stenosis of the larynx with certainty, for it demands speedy therapeutic interference.

Treatment.—In the beginning of the affection we may try to reduce the inflammation by the internal and external application of ice or by leeches. The pain may be alleviated by injections of morphin or painting with cocain. If stenosis of the larynx occurs, surgical interference is usually necessary, for only in very rare cases do we see the abscess open of itself with a subsidence of the dangerous symptoms. In the majority of cases the patient can be saved from suffocation only by the timely performance of tracheotomy or laryngotomy. The laryngeal abscess has been repeatedly opened internally by laryngologists with favorable results. If a chronic stenosis of the larynx remains after a favorable termination of the disease, either the patient must wear a tracheal cannula all his life, or the attempt may be made to dilate the stenosis gradually by the methods referred to in the preceding chapter.

CHAPTER IV

ŒDEMA OF THE GLOTTIS

The practical importance of the subject demands a brief special description of œdema of the glottis, by which we mean œdema of the entrance of the larynx, especially of the ary-epiglottic ligaments. We have already learned to recognize laryngeal perichondritis as one of its most frequent causes. In less deeply seated inflammations in the larynx and its neighborhood, however, œdema of the glottis may sometimes develop as a dangerous complication, especially in cases of laryngitis occurring in the course of severe acute diseases, such as typhoid, smallpox, or erysipelas, or in inflammations of the larynx arising from severe mechanical or chemical irritation, as from hot steam or corrosive substances, or from wounds of the larynx, or, finally, from foreign bodies in the larynx. The collateral œdema in angina Ludovici, in intense inflammations of the parotid gland, or the tonsils, etc., may in rare cases extend to the ary-epiglottic ligaments. Finally, œdema of the glottis occurs in rare cases as a complication of general œdema of the body, as a result of Bright's disease, disease of the heart, emphysema of the lungs, etc. Œdema of the glottis, without extensive œdema elsewhere, has been repeatedly observed to come on quite suddenly, especially in acute and chronic nephritis.

The chief symptom of œdema of the glottis is dyspnœa, which comes on as a result of the stenosis of the entrance of the larynx, and is sometimes extreme. At first this is chiefly on inspiration, but it soon comes on with expiration also. Respiration, especially inspiration, is accompanied by a loud laryngeal stridor. As a result of the incomplete entrance of the air, the efforts at inspiration cause depression of the root of the neck, the epigastrium,

and the sides of the thorax. We see with the laryngoscope, if the examination be successful, an œdematous swelling of the ary-epiglottic ligaments, and often a swelling of the epiglottis and the false vocal cords. Sometimes we succeed in feeling the swollen parts with the finger.

If the dyspnœa reaches a degree which threatens life, an operation is the only thing which can afford relief. Laryngologists attempt to reduce the swelling by long incisions in the œdematous parts. If this does not succeed, tracheotomy must be performed. If the immediate danger to life is thus averted, further treatment should be directed to the disease which has given rise to the œdema.

CHAPTER V

TUBERCULOSIS OF THE LARYNX

(Laryngeal Phthisis. Consumption of the Larynx)

Ætiology.—Since tuberculosis of the larynx is in most cases combined with tuberculosis of other organs, especially of the lungs, we must refer to the description of tuberculosis of the lungs for the general ætiology and pathology of the disease. A particular description of the special appearances in laryngeal tuberculosis is, however, justifiable, because tuberculosis may at times begin in the larynx and may remain isolated there, at least for a time; and, furthermore, in many cases of laryngeal tuberculosis, which are evidently combined with pulmonary tuberculosis, the laryngeal symptoms are predominant in the clinical picture of the disease. Many physicians have, wrongly as we think, disputed the fact that tuberculosis can begin in the larynx. Clinical experience not infrequently teaches us that men, who have been apparently in good health, are attacked with hoarseness, the disease being at first thought to be a common laryngitis, but at last, by its course, proving to be tuberculosis. In spite of the most careful examination, there are not to be found at first the slightest physical signs of disease in the lungs, and not till later do the manifest signs of a pulmonary tuberculosis succeed the symptoms of a laryngeal affection. In such cases it seems to us an affectation to claim that there is a primary pulmonary tuberculosis which could not be made out at first. Everything is much more in favor of the opinion that tubercle bacilli may sometimes, if not frequently, first fix upon the larynx, excite the first symptoms of tuberculosis there, and then attack the lungs.

In the great majority of cases of laryngeal tuberculosis the symptoms are developed, of course, secondarily in the course of chronic pulmonary phthisis. We shall see that in most cases the disease of the larynx is to be considered as the result of an infection of the mucous membrane of the larynx by the tuberculous sputum which passes over it. More rarely the infectious material seems to reach the larynx by way of the lymph or blood vessels. In favor of this theory we may mention that men of great experience (Schrötter and others) have stated that, when the laryngeal trouble was particularly marked on one side, that side was very often affected which corresponded to the affected lung. In about one fourth of all cases of pulmonary

tuberculosis this complication occurs, if we include all the mild laryngeal cases. Marked and extensive tuberculosis of the larynx is much rarer, however.

Pathological Anatomy.—Tuberculosis of the laryngeal mucous membrane begins, like tuberculosis of other mucous membranes, with the formation of small subepithelial nodules, which soon become cheesy, break down, and form small ulcers. In the further course of the disease there is very often a more extensive tubercular infiltration, which gives rise on the one hand to irregular thickening of the mucous membrane, and on the other to deep ulceration. The tubercular infiltration is most frequently situated in the interarytænoid space, on the false or true cords (at first on one side), and on the epiglottis. The tuberculous ulceration usually develops later in one of these places. The ulceration may extend from the epiglottis to the neighboring parts of the tongue. In severe cases we often find a marked collateral œdema in the surrounding parts accompanying the inflammation, and sometimes the tubercular perichondritis which has already been described.

The rest of the laryngeal mucous membrane (especially the vocal cords) not involved in the specific tubercular process is usually the seat of a simple catarrh.

Clinical Symptoms.—In the beginning of tuberculosis of the larynx the laryngoscope usually shows nothing but the appearances of a simple catarrh, since the primary miliary nodules of tuberculosis are usually hard to make out. If the catarrhal symptoms be only on one side, we should always suspect tuberculosis. In the later stages, however, most of the special details of the destructive tubercular process, described above (ulcers, infiltration, etc.), can be very satisfactorily made out with the laryngoscope. The infiltration and ulcers are either chiefly on one or on both vocal cords or on the posterior wall of the larynx, or mainly in the neighborhood of one arytænoid cartilage, or the whole epiglottis is irregularly ulcerated on its edge. Single tubercular nodules are often plainly to be seen. In severe cases almost the whole upper portion of the larynx appears like an ulcerated surface covered with mucus and pus. In laryngeal tuberculosis, however, the soft palate usually looks very pale and anæmic. The redness and swelling of the affected parts are more distinct in the living subject than in the cadaver; but the autopsy often shows a far greater extension of the tubercular disease than we can determine by the laryngoscope.

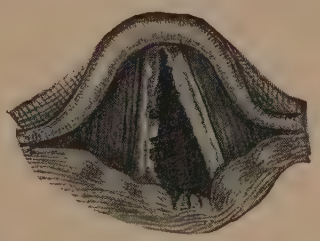

Fig. 31.—Beginning laryngeal tuberculosis (from Sahli).

The other clinical symptoms of tuberculosis of the larynx vary very much with the extent and intensity of the process. Sometimes they consist merely in moderate roughness and hoarseness of the voice, but in other cases they increase to the most painful condition which is ever seen in any variety of tuberculosis. This is especially apt to be the case if the ulceration involves the epiglottis and the arytænoid cartilages. Swallowing is then extremely painful, so that the nutrition is very often impaired, and painful attacks of coughing frequently occur. A practiced ear can often distinguish at once the

hoarse sound of a "laryngeal cough" from the ordinary pulmonary cough. If severe ulcerations attack the vocal cords, and their free mobility is affected to a marked degree, the hoarseness increases, and finally reaches a complete aphonia. Death finally occurs from general inanition, or, exceptionally, from œdema of the glottis.

Diagnosis.—The diagnosis of tuberculosis of the larynx is not difficult if pulmonary phthisis is already known to be present. When attention has been called to it from the onset of hoarseness or from some disturbance in swallowing, we recognize the character and seat of the changes by the aid of the laryngoscope. The diagnosis, however, may present much difficulty in cases where we are not sure that an affection of the lungs coexists. As has been said, the symptoms at first are not unlike those of a simple catarrh, and the suspicion of the existence of tuberculosis is first aroused from the stubbornness of the disease, the condition of the patient, the knowledge of some inherited taint, the onset of fever, and the remarkable anæmia and emaciation. With the changes in the larynx which have been described, without evidence of pulmonary tuberculosis, the distinction between tuberculosis and syphilis may be very difficult. In syphilis of the larynx, however, we find that coexisting changes in the pharynx are much commoner than in tuberculosis, and the cicatricial formation which is usually visible furnishes, besides, a very characteristic evidence of syphilis. The diagnosis of tuberculosis of the larynx, however, is made perfectly certain in all doubtful cases by the discovery of tubercle bacilli in the patient's expectoration or in the secretion from the ulcer, which often can be easily obtained by the aid of a fine laryngeal brush. In regard to the laryngoscopic appearances, we may also say that a thickening and infiltration of the epiglottis with a partial ulceration of the same is an appearance which is almost exclusively confined to tuberculosis. The same is true with regard to a marked projecting infiltration of the interarytænoid region. In doubtful cases it may aid the diagnosis to inject tuberculin, or, on the other hand, to watch the results of antisyphilitic treatment.

Treatment.—For the constitutional treatment of tuberculosis of the larynx the reader is referred to the description of pulmonary tuberculosis. We shall here discuss merely the local treatment. This is in the milder forms the same as for simple laryngeal catarrh. There is no doubt that even genuine tuberculous ulceration of the larynx may be healed. Nevertheless, permanent cures of this sort are exceptional. Of course very much depends upon the coexisting state of the lungs. For local treatment insufflations of iodoform and iodol were for a time strongly recommended, but they are now for the most part abandoned. Cauterizing the tuberculous ulcers with nitrate of silver does more harm than good. We sometimes see better results from blowing powdered boric acid on the ulcers or from a twenty-per-cent solution of menthol in oil. At present lactic acid is most recommended—cauterizing the ulcers with a solution of increasing strength (twenty to eighty per cent), at first daily, and later, after the ulcers have been cleaned off, less frequently. Preliminary cocainization of the parts to be cauterized is always necessary. Ulcers presenting exuberant granulations should be scraped with specially constructed spoons and curettes.

If we were to give a general estimate of the local treatment of laryngeal tuberculosis we should have to admit that its success has not been great. We

do not deny that skill and perseverance may be of much service, but only too often do we become discouraged by the failure of the most careful local treatment, and in many cases cauterization of the larynx is only a useless distress to the patient.

With incipient disease there may be considerable improvement and even recovery without any local treatment if the larynx be spared any exertion and given complete rest and if, at the same time, careful general treatment be instituted. The most we need to use is the atomizer or nebulizer with simple salt solution, alkaline waters, etc. We have also found inhalations of a weak carbolic solution, and especially of vapor of Peruvian balsam (bals. Peruviani, ʒijss. [gm. 10]; spiritus vini, ʒj ♏xv [gm. 5], 20 drops in hot water, to be inhaled through a funnel three times daily) of service. In advanced cases, where pronounced pulmonary tuberculosis coexists, we must content ourselves with a purely palliative treatment. The constant use of cracked ice, and especially a lavish employment of narcotics, are of most service in relieving the pain and the difficulty in swallowing. Subcutaneous injections of morphin a quarter of an hour before a meal often afford great relief. We have seen very good results from cocain. If we paint the ulcerated mucous membrane at the entrance of the larynx with a ten- or twenty-per-cent solution of cocain, in a few minutes such an anæsthesia of the affected parts ensues that swallowing may take place without any pain. The following formula may be used:

℞ Cocainæ muriatis	.....	gr. xv–xxx,	gm. 1.0–2.0;
Alcohol	.....	ʒss.,	2.0 c.c.;
Aquæ destillatæ	.....	ʒij,	8.0 c.c.
M.			

Unfortunately the action of cocain is very transitory, so that the painting must be frequently repeated. Insufflations of orthoform or of morphin in powdered form (gr. $\frac{1}{6}$ [gm. 0.01], morphin with gr. iijss. [gm. 0.2], pulv. gummosus [1]) are worthy of trial. With a severe laryngeal cough and irritation relief may also be obtained by inhalations of a two- to three-per-cent solution of bromid of potassium or a three- to ten-per-cent solution of bitter-almond water.

If a pronounced stenosis of the larynx develop, tracheotomy is indicated. Many recent observations seem to show that tracheotomy may also be of service in marked laryngeal tuberculosis. When the laryngeal respiration is shut off the tubercular lesions seem to improve faster.

In cases of pronounced but comparatively limited laryngeal tuberculosis, without much implication of the lungs, the attempt has been made, after a preliminary laryngotomy, to remove the diseased parts as completely as possible by surgical means. We still lack extended practical experience in this matter, but further trials of the operation seem to me to be very desirable.

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[1] P. G. Gumacacia 15 parts, licorice root 10, sugar 5.

CHAPTER VI

PARALYSES OF THE LARYNGEAL MUSCLES

PARALYSES IN THE DISTRIBUTION OF THE SUPERIOR LARYNGEAL NERVE

The superior laryngeal nerve, arising from the vagus, is the sensory nerve for the mucous membrane of the upper portion of the larynx down to the glottis, and also for the mucous membrane of the epiglottis and its neighborhood. Besides this, it also supplies motor fibers to the cricothyroid muscle, probably from the accessory nerve. Clinical experience renders it probable that the superior laryngeal nerve also supplies the depressors of the epiglottis, the thyro-epiglottideus, and the arytæno-epiglottidei muscles, and perhaps also the arytænoideus muscle. The last three muscles mentioned, however, perhaps derive some motor fibers from the recurrent nerve also (the inferior laryngeal nerve).

Paralysis of the cricothyroid muscles and of the depressors of the epiglottis is seen most frequently after recovery from diphtheria. It is usually a part of a more extensive paralysis, and, in addition, is sometimes associated with anæsthesia of those portions of the mucous membrane which, as we have seen, derive their sensory fibers from the superior laryngeal nerve (von Ziemssen). Choking is therefore easy and there is danger of inhalation pneumonia.

Paralysis of the thyro-epiglottideus and the arytæno-epiglottidei muscles is recognized by the immobility and the erect position of the epiglottis, which is directed toward the back of the tongue.

Paralysis of the cricothyroid muscles makes the voice rough, and especially renders the production of high tones impossible, since for this purpose we need the action of this muscle as a tensor of the vocal cords by approximating the cricoid and thyroid cartilages. The detection of this paralysis by the laryngoscope is extremely difficult. Its chief signs are a concavity of the edges of the vocal cords, a lack of visible vibration in them, and perhaps, in unilateral paralysis, a higher position of the vocal cord on the sound side.

For paralysis of the arytænoideus muscle, *vide infra.*

PARALYSES IN THE DISTRIBUTION OF THE INFERIOR LARYNGEAL OR RECURRENT NERVE

The recurrent nerve supplies with sensory fibers the mucous membrane of the inferior cavity of the larynx below the glottis, and it is the motor nerve for all the laryngeal muscles except the cricothyroid, and except possibly the depressors of the epiglottis (*vide supra*). The muscles innervated by it are arranged according to their function in the three following groups:

a. The openers of the glottis—the posterior crico-arytænoid muscles.

b. The closers of the glottis—the lateral crico-arytænoids and the arytænoideus (transverse and oblique).

c. The tensors of the vocal cords—the thyro-arytænoids, which act usually as closers of the glottis, but which very often produce the fine differences in tension in the vocal cords which are necessary in singing and in modulations of speech. They accordingly have the same task as the coarser-working crico-thyroid muscles, which are innervated by the superior laryngeal nerve.

The motor fibers for all these muscles have their special origin in the accessory nerve, from which they pass into the trunk of the vagus, and from this into the laryngeal nerves.

Most of the paralyses of the recurrent nerve are of peripheral origin. Except in the pure muscular pareses (*vide supra*), which arise not infrequently in the course of other laryngeal affections, peripheral paralyses of the vocal cords occur with the greatest relative frequency from an abnormal pressure on the trunk of the recurrent nerve, especially in aneurism of the arch of the aorta, which may cause a left-sided paralysis. Tumors of the bronchial glands, cancer of the œsophagus, thyroid or mediastinal tumors, and, in rare cases, even large pericardial effusions, may also cause a paralysis of the recurrent on one side. Paralyses on the right side are seen quite frequently in contractions at the apex of the right lung and in rare cases of aneurism of the subclavian artery. The paralyses of the laryngeal muscles, which are sometimes met with after recovery from diphtheria (*q. v.*), also belong to the peripheral paralyses of the recurrent nerve, and their cause is to be found in a degeneration of the branches of the affected nerves. In other cases the paralysis of the recurrent nerve is due to an affection of its fibers in the vagus or even in the accessorius. Excluding certain injuries from operations, these affections are usually due to new growths which cause a paralysis of conduction. Paralyses of the recurrent nerve also arise from affections of the nucleus of the accessory nerve in diseases of the medulla, in the different forms of acute bulbar paralysis, in chronic bulbar paralysis, in multiple sclerosis, etc. The frequent hysterical paralyses affecting the closers of the glottis and the tensors of the vocal cords are to be regarded as central disturbances of innervation (see the chapter on hysteria).

1. **Complete Paralysis of the Recurrent Nerve.**—Paralysis of all the laryngeal muscles supplied by the recurrent nerve occurs quite frequently in the pressure paralysis of the trunk of the recurrent, or of its fibers in the vagus. With the laryngoscope (see Fig. 32) we find the vocal cord on the paralyzed side in a middle position, often falsely called a "cadaveric position," and completely motionless on respiration, and also on phonation. The arytænoid cartilage on the paralyzed side is often turned inward. On phonation as strongly as possible, the vocal cord on the sound side passes beyond the median line, the arytænoid cartilage also crosses the line, and consequently the glottis is put into an oblique position. The other symptoms are sometimes so slight that without a laryngoscopic examination we do not even think of a paralysis. The voice, however, is usually not pure; it often breaks into a falsetto, and the patient is easily tired by speaking. With

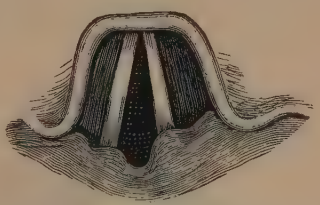

Fig. 32.—(From Ziemssen.) Position on inspiration in paralysis of the left vocal cord, or paralysis of conduction in the recurrent nerve.

complete bilateral paralysis of the recurrent nerve, which is very rare, we find both vocal cords motionless in a middle position. Complete aphonia exists, and it is impossible to cough, since in coughing we have to make at first a complete closure of the glottis. There is no dyspnœa, however, if the patient keeps quiet.

It is an interesting fact, first reported by Rosenbach, that in incomplete recurrent paralysis almost invariably the abductors of the vocal cords (the openers of the glottis) are alone paralyzed, whence the cords remain in adduction. Only with the further advance of the disease does complete recurrent paralysis occur; then the adductors also become paralyzed, and the vocal cords become completely motionless, and assume the middle, so-called cadaveric position.

2. **Paralysis of the Dilators of the Glottis, the Posterior Crico-arytænoid Muscles.**—Bilateral paralysis of these muscles is quite a rare phenomenon, but clinically it is of the utmost importance, since it results in a condition of most marked inspiratory dyspnœa. Neuritic changes, central diseases (tabes, multiple sclerosis, etc.), cancer of the œsophagus, etc., may lead to paralysis of the posterior muscles. In many cases the cause is obscure. The sequelæ of the paralysis usually develop slowly and gradually. The affection may last for years. The greatest impairment of breathing probably occurs when the vocal cords are fixed in adduction by the contracture of the antagonistic closers of the glottis. The dyspnœa may increase, especially from external causes, to severe attacks of suffocation, and tracheotomy is frequently necessary. The respiration is affected in such a way that inspiration only is difficult, protracted, and noisy, while expiration is free and unhindered. This depends on a valvelike action of the vocal cords. They are drawn together by the dilatation of the thorax on inspiration, while the current of air in expiration easily pushes them aside. Phonation is usually entirely undisturbed. With the laryngoscope (see Fig. 33) we find the glottis changed to a small slit, which grows narrower instead of wider on inspiration. The prognosis is usually unfavorable. Only in the hysterical can these apparently severe conditions appear and disappear again in a short time.

In unilateral paralysis of the posterior crico-arytænoid muscle there is, as a rule, no marked dyspnœa. The voice is somewhat hoarse, and with the laryngoscope we can see that the paralyzed vocal cord does not deviate outward on inspiration.

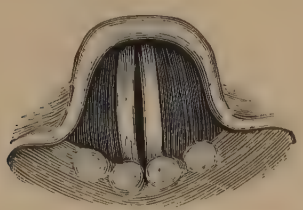

Fig. 33.—(From Ziemssen.) Complete bilateral paralysis of the posticus at the moment of inspiration.

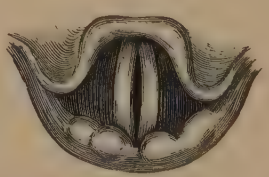

Fig. 34.—(From Ziemssen.) Paralysis of both internal thyro-arytænoid muscles in the course of an acute laryngitis.

3. **Paralysis of the Thyro-arytænoid Muscles.**—The paralysis or paresis of these muscles, which run into the vocal cords, and which are their

chief tensors, is one of the most frequent of the paralyses of the laryngeal muscles. It occurs especially in acute and chronic catarrh of the laryngeal mucous membrane, and is often the chief cause of the accompanying hoarseness. It also frequently develops as the result of an habitual strain of the voice in singers and public speakers, and it is one of the commonest causes of hysterical aphonia.

Paralysis of the thyro-arytænoid muscles may be bilateral or unilateral. It is frequently associated with a paresis of the other closers of the glottis, the arytænoidei and the cricothyroid muscles. With the laryngoscope (see Fig. 34), in the ordinary bilateral paresis of the thyro-arytænoid muscles, we see that on phonation the glottis does not close completely, but that an oval space is left between the vocal cords.

In unilateral paralysis the affected cord shows a concavity of its edge. The voice is always more or less hoarse and weak, and the speech is strained.

In many cases, after a cure of the original catarrh, a complete recovery from the paralysis may follow by taking good care of the voice. Hysterical paralyses are diagnosticated by their sudden disappearance and reappearance, usually after some psychical disturbance. They are quite common in children of the age of ten to fourteen years, especially in girls. (See chapter on Hysteria.)

4. **Paralysis of the Arytænoideus Muscle.**—Paralysis of this muscle is rarely an isolated phenomenon. It is sometimes seen in laryngeal catarrh or in hysterical aphonia. The voice is quite hoarse, and with the laryngoscope (see Fig. 35) we find on phonation that the whole anterior part of the vocal

Fig. 35.—(From Ziemssen.) Paralysis of the arytænoideus in acute laryngitis.

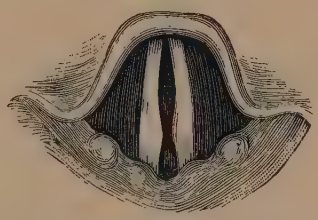

Fig. 36.—(From Ziemssen.) Bilateral paralysis of the thyro-arytænoids combined with paresis of the arytænoideus.

cords closes well, but that the cartilaginous glottis remains open as a triangular gap on account of the imperfect motion of the arytænoid cartilages toward each other. When the thyro-arytænoids are paralyzed with the arytænoideus, the glottis shows on phonation a narrow hour-glass opening (see Fig. 36). Both the anterior and the posterior portions of the glottis fail to close, while the vocal processes take their usual median position on phonation from the normal turning of the arytænoid cartilages inward by the action of the lateral crico-arytænoid muscles.

5. **Paralysis of the Lateral Crico-arytænoid Muscles.**—As an uncomplicated condition, paralysis of these muscles has never been observed with certainty. Some cases of a complete and simultaneous paralysis of all the closers of the glottis have been described, however, in which the vocal cords are immovable laterally and the glottis remains abnormally wide open.

TREATMENT

We may expect success from the treatment of paralysis of the vocal cords only when the primary disease is capable of cure. If catarrhal or other diseases of the larynx coexist, we must first treat these by the methods already mentioned. Paralysis from the compression of tumors, etc., may be relieved in rare cases by extirpation, or by partial resolution of the tumors when of strumous origin. In catarrhal, diphtheritic, and the so-called "rheumatic" pareses—that is, those which occur without any assignable cause—and also in all hysterical aphonias, electricity often works very well. Von Ziemssen has made electrodes for the endolaryngeal stimulation of single muscles, but external galvanization is usually sufficient in practice. In hysterical aphonia the chief task is to accustom the patient again to the necessary and proper voluntary innervation. We usually accomplish our purpose most rapidly by commanding the patient to cough and then say "Ah," while we faradize or galvanize the neck. It is often of advantage to reverse the current. If the patient succeeds in saying "Ah" once, the voice usually returns very quickly. Internally we may try subcutaneous injections of strychnin in doses of gr. $\frac{1}{20}$ to gr. $\frac{1}{6}$ daily (gm. 0.003 to 0.01).

CHAPTER VII

SPASM OF THE GLOTTIS

(Laryngismus Stridulus. Millar's Asthma. Thymic Asthma)

Ætiology.—Spasm of the glottis is a disease which occurs almost exclusively in children under three years of age, and consists of attacks of spasmodic closure of the glottis, and consequently of most severe dyspnœa. Boys are more frequently attacked by this disease than girls, but the cause of this is wholly unknown. The old name of thymic asthma arose from the idea that the attacks were due to an increase in the size of the thymus gland, but this opinion is wholly unfounded. The relation between spasm of the glottis and rachitis is remarkable, but it is unexplained. Nearly two thirds of all the children who suffer from spasm of the glottis are rachitic, but the opinion which was once held that spasm of the glottis has a special relation to the rachitic craniotabes is not clearly proved. The fact that it is often combined with eclampsia, in that the attacks of spasm of the glottis are aggravated by eclamptic attacks, and that the two alternate with each other, is an argument in favor of a central origin for the disease. In the cases which come on, as they often do, at the time of dentition, we think it possible to assume a reflex origin for the spasm, just as we may in those cases which seem to follow a laryngitis due to taking cold.

Symptomatology.—The single attacks usually come on suddenly by day or by night, either without any cause or from some external influence, such as crying, swallowing fluid, or some psychical disturbance. They usually begin with a deep inspiration, followed by complete cessation of respiration. The child becomes pale, cyanotic, looks anxiously about, rolls his eyes, and

makes strained and labored efforts at respiration. In severe cases there is a temporary loss of consciousness, and tonic and clonic spasms in the muscles of the extremities and the trunk, as has been mentioned. The attack lasts from some seconds up to two minutes. In very severe cases the attack may be immediately fatal. As a rule, however, the spasm passes off, deep, noisy inspirations follow, and in a short time the child is completely well. The severity of the attacks varies, moreover, in different cases, and it varies very markedly, too, in the same child. Sometimes we have only one attack or a small number of them, while in other children they may come on ten or twenty times a day, and even oftener, and may last with varying intensity for months. If the child reaches his third year the disease almost always disappears, but quite a large number of the children who suffer from spasm of the glottis die before that age, either in the attack itself or from other affections.

Genuine spasm of the glottis hardly ever occurs in adults, but similar attacks, which, of course, have quite a different significance, are sometimes observed in hysteria.

Treatment.—The treatment must be especially directed to the child's general condition. The child is usually pale and emaciated, and if we succeed in improving its nutrition with iron and cod-liver oil, the attacks become less frequent, and milder, and finally may wholly disappear. If rachitis exists, we should first of all try phosphorus (1 part in 10,000 of cod-liver oil). The child should also be kept in moderately warm air and guarded from any exposure to cold. Internal remedies to prevent the recurrence of the attacks are very uncertain in their action. We may employ 1 or 2 gr. (gm. 0.05 to 0.10) of chloral hydrate in solution every hour or so; bromid of potassium, 10 to 30 gr. daily (gm. 0.5 to 2.0); extract of belladonna, etc.

In the attack itself the child must be raised up. The face should be sprinkled with water, or, if the attack be of long duration, a cool shower bath should be given. Friction should be applied to the skin, aided by mustard, or a mustard plaster applied to the chest and calves. If the attacks are very frequent and intense, we must use narcotics, either inhalations of chloroform or subcutaneous injections of morphin, with care, in doses, for a child, of $\frac{1}{60}$ to $\frac{1}{12}$ of a gr. (gm. 0.001 to 0.005).

CHAPTER VIII

NEW GROWTHS IN THE LARYNX

Since new growths in the larynx are of interest mainly to specialists and surgeons, we will here glance but briefly at them. We must remember especially, however, that they can be recognized only by the aid of the laryngoscope. It unfortunately often happens that a patient is treated for a long time without success for a "chronic laryngeal catarrh," when the laryngoscope finally shows that a new growth is the cause of the hoarseness. It is of especial importance, however, to make a diagnosis as early as possible, particularly in carcinoma, since the earlier the operation is done the better is the chance for success (*vide infra*).

A. BENIGNANT GROWTHS IN THE LARYNX

1. **Papilloma.**—Papilloma is one of the commonest new growths in the larynx. It forms glandular, cauliflower-like excrescences, which are usually situated on the anterior part of the vocal cords, rarely on the false cords. The base of the swelling is broad or pediculated. The growths are often multiple. We do not know the special cause of their origin. It is worthy of note that papilloma, like other benignant growths in the larynx, is seen much more frequently in men than in women.

2. **Fibroma.**—Fibroma in the larynx is comparatively common. The pediculated tumors known as "laryngeal polypi" are usually fibromata. They are generally situated on the vocal cords and form whitish or reddish-

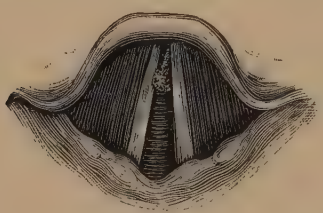

Fig. 37.

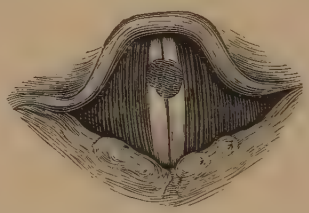

Fig. 38.

Figs. 37 and 38.—(From Ziemssen.) Pediculated fibromata.

brown swellings, from the size of a pea to that of a cherry, vascular and dense or soft (see Figs. 37 and 38). There is no evidence that persons who use their voices very much are especially liable to the formation of fibromata.

3. **Cysts and Polypi.**—Cysts and "mucous polypi" rarely occur. They are probably due to the retention of the secretion in a mucous gland from the stoppage of its orifice. We find them in the ventricles of Morgagni, on the epiglottis, etc. In a very few cases we also see in the larynx lipomata, myxomata, separated portions of thyroid-gland tissue which have begun to grow, enchondromata, etc.

The symptoms which are excited by benignant tumors in the larynx depend partly upon the situation and partly upon the size of the new growth. Small polypi may exist wholly without symptoms, and they are found only by chance on laryngoscopic investigation. Usually, however, the presence of disturbances of phonation (hoarseness, a marked change in pitch, secondary tones), persistent cough, or respiratory disturbances when the tumor is a large one, are the symptoms which give occasion for an examination. Abnormal sensations in the larynx are by no means the rule, and pain is wholly absent.

B. MALIGNANT GROWTHS. CARCINOMA OF THE LARYNX

Sarcomata, arising from the true or false vocal cords, are very rare. The only malignant new growth of the larynx of great practical importance is carcinoma.

Carcinomata develop usually in old people, either primarily in the

larynx or secondarily from affection of the neighboring organs. In the first case the vocal cords or the ventricles of Morgagni are the points most frequently attacked. An extension of the disease to the larynx is seen especially in cancer of the tongue or pharynx, rarely in cancer of the œsophagus.

The symptoms of cancer of the larynx develop slowly. Hoarseness, dysphagia, pains in the larynx often shooting up into one ear or the temporal region, the appearance of respiratory symptoms, and finally, but only in the later stages, as a rule, the signs of general weakness and emaciation which are seen in almost all forms of carcinoma, form the picture of the disease. Enlargement of the cervical lymph-glands is sometimes an early symptom, but it may be wholly lacking. The diagnosis is possible only by the aid of the laryngoscope. Besides this, a digital examination may at times be of diagnostic value by the detection of the characteristic hardness about the entrance or in the neighborhood of the larynx. A general description of the laryngoscopic appearances cannot be given on account of the diverse character of the cases. We see the uneven, injected new growth, covered with mucus and often ulcerated, and besides this at times the secondary appearances of catarrh, a developing perichondritis, etc. In many instances the diagnosis is quite easy, but in other cases protracted observation is required. It may be difficult, however, at times, to distinguish it from tuberculosis or from syphilis. We may be aided in such cases by a search for tubercle bacilli, by tuberculin injections, or by the results of antisyphilitic treatment. All the other organs of the patient therefore must always be carefully examined. In doubtful cases it is proper to remove a bit of the endolaryngeal growth and examine it histologically.

TREATMENT

Surgical treatment is the only one for all laryngeal new growths. We must refer to the special works for the details. Laryngologists have devised numerous instruments for the removal of benignant polypi, by which, under the guidance of the laryngoscope, the new growth is cut, snared, crushed, or torn off. The performance of the operation is made much easier by inducing local anæsthesia of the laryngeal mucous membrane by painting with cocain. Nevertheless, we firmly believe that in all severe cases the "endolaryngeal" operations should be superseded by laryngotomy. Carcinoma of the larynx can be cured only through removal of the tumor by laryngotomy (splitting of the larynx) or by total extirpation of the larynx. Laryngotomy is comparatively free from danger, while total extirpation has met with permanent success as yet in but few instances. If surgical interference is no longer practicable, we can endeavor merely to mitigate the suffering of the patient by means of morphin, cocain, and other narcotics.

SECTION III

Diseases of the Trachea and the Bronchi

CHAPTER I

ACUTE CATARRH OF THE TRACHEA AND THE BRONCHI

(Tracheitis and Acute Catarrhal Bronchitis)

Ætiology.—Acute catarrh of the larger air-passages, of the trachea, and larger bronchi, is a frequent disease, and it may often arise from taking cold. It is conceivable that the inhalation of cold, damp air sometimes directly affects the mucous membrane of the upper air-passages. Bronchial catarrh is very often associated with a coincident catarrh of the larynx, and more rarely of the pharynx. In the ordinary mild forms the catarrh is usually confined to the trachea and the first large branches of the bronchi, while the finer bronchi remain healthy.

More intense inflammation of the bronchial mucous membrane is the result of active mechanical or chemical irritation. A severe bronchitis develops after the inhalation of noxious gases, nitrous and sulphurous oxids, chlorin, bromin, etc., as is often observed in operatives. The inhalation of smoke and dust, especially vegetable dust, works in the same injurious fashion, and the followers of many trades and employments, such as millers, wool workers, colliers, etc., are especially subject to disease from this cause. In this form of bronchitis the catarrh often extends to the finer bronchi.

The bronchitis which develops in the course of other acute and chronic disease is still commoner than the primary forms already mentioned. It is often due to infectious causes, such as certain infectious diseases, especially measles, whooping cough, and influenza. In these diseases bronchitis is one of the most constant local affections, and is probably dependent immediately upon the primary infection. Bronchitis, however, develops secondarily in most of the other acute infectious diseases, and is due largely to the inhalation of noxious substances from the upper part of the air-passages. This is the explanation of the bronchitis in diphtheritic processes in the pharynx and larynx, in so far as it does not depend upon a direct extension of the disease, and also of the bronchitis in smallpox, etc. Bronchitis may also be met with in all other forms of severe disease, because retention of secretion, putrefactive processes, inflammation, thrush, etc., occur in the cavity of the mouth and pharynx, and from them chemical or organic irritants may easily be inhaled into the bronchi. The imperfect expectoration in all severe diseases is a still more harmful factor than this inhalation. The secretion remains in the bronchi, processes of decomposition arise in the stagnating mucus, bacteria collect and lead to a bronchitis, and finally to the lobular pneumonia which is so often found (*vide infra*). The lessened power of resisting injurious influences, produced in the mucous membrane by severe bodily disease, also facilitates the development of catarrhal inflammation. In

all disease in which deglutition is affected (as in paralysis of the epiglottis or of the pharyngeal muscles), and in all diseases attended by frequent vomiting or choking (cancer of the œsophagus, etc.), secondary bronchitis with its sequelæ may ensue from the inhalation of small particles of easily decomposable food.

We do not know how far we may claim that infectious agents act as a cause of primary bronchitis, yet it is not improbable that many cases have such an ætiology. It is especially likely that many cases of bronchitis due to "catching cold" really have something infectious about them, and that the preceding exposure to cold has merely lowered the natural powers of resistance, and thus permitted, or at any rate promoted, infection. This infection is probably not due to any special specific bacteria but to the ordinary germs of inflammation (staphylococci, etc.).

Finally, we must mention that acute bronchitis is sometimes merely an exacerbation of a previous chronic bronchitis. We will return later to this important and not uncommon process.

The predisposition to acute bronchitis varies in different persons. We do not know definitely on what ground such an increased predisposition to bronchial disease rests, nor why we meet with it sometimes in the weak and anæmic, and at other times in so-called "full-blooded" persons. Bronchitis is more frequent in children and old people than in those in middle life. Most of the cases occur in the spring and autumn.

Symptoms.—Pain in the chest may be present in some cases of simple catarrhal bronchitis, but usually only in a moderate degree. In severe tracheitis patients often have a painful feeling of soreness in the neck and behind the upper part of the sternum, and this is increased on coughing. The mucous membrane of the bronchi, apparently, has no nerve fibers which are sensitive to pain, and the pains in the chest which are often present in bronchitis are, as a rule, muscular pains in the intercostal muscles, or the insertion of the diaphragm, due to the severe paroxysms of coughing.

Cough is one of the most constant symptoms of bronchitis, and by it usually the attention of the patient or of the physician is first called to the existing thoracic affection. The cough may, of course, be due to a laryngitis, if that is also present. There is no doubt, however, that a cough may be excited in a reflex manner from the mucous membrane of the trachea and of the larger as well as of the finer bronchi. Experiments have shown that the point of bifurcation of the trachea is especially irritable, and many severe paroxysms of coughing may be due to an irritation of this very spot from the accumulation of secretion. The intensity of the cough, moreover, is very different in individual cases, which is due in part to the degree and extent of the bronchitis and in part to the individual irritability of the person affected.

The expectoration consists of the secretion and exudation from the inflamed mucous membrane. Its abundance and consistence vary considerably in the different cases. We distinguish a catarrh with an abundant secretion from the so-called "dry catarrh." In the latter only a little viscid sputum is expectorated, but in the former the expectoration is more abundant and mucopurulent, or seromucous, a thin fluid separating into layers on standing (*vide infra*). Very often in the beginning of the disease the expectoration is scanty and viscid—the *sputum crudum* of the old physicians; and later

it becomes more abundant, more fluid, and more purulent—the *sputum coctum*. In catarrh of the finer bronchi the expectoration may contain little tough mucous or mucopurulent casts of the bronchi. It may be stated in general that the characteristic of simple bronchitic sputum is the mucous admixture in contrast to the purulent or seropurulent sputum from phthisical cavities, etc. We may readily recognize the mucous contents of the sputum by the tenacity with which it clings to the bottom of the sputum cup when the cup is tipped. A simple catarrhal expectoration shows nothing peculiar under the microscope. The pus corpuscles are often swollen, and show more or less marked fatty degeneration. A slight admixture of blood may occasionally be present in severe bronchitis, but it usually has no special significance, being at times merely the result of severe fits of coughing. A more marked and persistent admixture of blood is seen in the catarrhal sputum of some cases of intense bronchitis in drunkards, so that we may even speak of a "hemorrhagic bronchitis."

Dyspnœa is usually entirely absent in simple mild bronchitis, but marked shortness of breath may be noticed in extensive catarrh of the finer bronchi.

Physical Examination.—We may obtain direct evidence of the condition of the tracheal mucous membrane, with due practice, by the laryngoscope. We see a reddening of the membrane, and sometimes an abnormal abundance of secretion on it, if there is a tracheitis. Other methods of physical examination are at our service for judging of the changes in the bronchi.

Inspection of the thorax shows nothing abnormal in the milder forms of bronchitis. The respiration is somewhat accelerated and the expiration prolonged in severe bronchitis, especially if the finer bronchi are affected. Percussion in uncomplicated bronchitis shows nothing abnormal in the pulmonary resonance. In extensive catarrh of the finer bronchi an acute dilatation of the lung is apt to occur, with a descent of the lower margin of the lung. Auscultation, too, shows nothing unusual in many cases of mild catarrh limited to the trachea and large bronchi, but in the cases in which the smaller bronchi are the seat of the catarrh and there is a marked accumulation of secretion in them, we hear, besides the vesicular respiration, the so-called rhonchi which almost wholly hide it. In dry bronchitis we speak of humming or buzzing sounds (sonorous rhonchi) or shrill, whistling sounds (sibilant rhonchi) according to their pitch. These sounds are probably due to stenosis, and are caused by the passage of the air through narrow portions of the bronchi. The narrowing occurs in part from the swelling of the mucous membrane, in part from the accumulation of secretion. The masses of secretion themselves, if they are set in vibration at the same time, may possibly take part in the production of the humming noises. If the amount of secretion collected in the bronchi is more abundant and of a more fluid consistence, it gives rise to "moist râles" on the passage of the air. These are distinguished as "coarse," "medium," or "fine moist râles," according as they occur in the larger or smaller bronchi.

Other symptoms of disease are often present besides those already mentioned as being due directly to the bronchitis. The general health is usually disturbed in a severe bronchial catarrh. The patient does not feel well, and has less appetite than usual. A moderate amount of fever is often present, especially toward evening. An increase of temperature above 102° or 103° F.

(39° C.) is rarely seen except in children. The patient sometimes complains of headache, which is increased by severe coughing. There can be a question of complications only when the inflammation extends to other parts of the respiratory mucous membrane. Mild bronchitis is often associated with coryza and laryngitis, while severe bronchitis may lead to catarrhal pneumonia (*vide infra*).

Forms of Bronchitis.—The separate forms of bronchitis are distinguished chiefly by the degree of extension of the catarrh.

1. The Milder Forms of Acute Bronchitis.—In most cases of simple primary bronchitis, as well as in many milder attacks of secondary bronchitis, the catarrh is limited to the mucous membrane of the larger bronchi. Exposure to cold and other injurious influences are frequent causes of the primary form. The symptoms are moderate. The cough, however, may be quite troublesome. Often fever is absent or but slight. Upon auscultation, particularly over the lower lobes, but sometimes over the entire lungs, are heard numerous rather coarse wheezing or rattling sounds, usually with some symmetry of distribution; but in many cases, as we have said, there may be nothing abnormal heard, so that the diagnosis will have to rest merely upon the subjective discomfort in the chest, the cough, and the expectoration. With proper care, simple primary bronchitis runs its course in a few days, or at the most in a few weeks, and ends in complete recovery. If the patient exposes himself recklessly, or the ætiological factors continue to be active, the disease may, however, grow worse, prove to be very tedious, and finally develop into chronic bronchitis.

2. The Severer Febrile Forms of Acute Bronchitis.—Sometimes acute bronchitis assumes a severer form, whether because the influences which give rise to it are unusually violent, or because it is due to some special and perhaps infectious cause. In such cases the symptoms are more marked, the bronchial râles more abundant, the general condition of the patient worse. Not infrequently there is fever for several days, or even for one or two weeks, the type being irregularly remittent, but the temperature is usually 102° or 103° F. (39° to 39.5° C.), although it may go higher. The expectoration is usually mucopurulent, but it often contains much pus; in other cases it may be sero-mucopurulent. The amount is then greater, and it forms distinct layers on standing in a beaker glass. The author has not infrequently noticed that there is more liability in the severer forms of acute bronchitis to have the disease mainly limited to one lobe, or at least to one lung, but we may have a severe diffuse bronchitis on both sides.

The ætiology of acute febrile bronchitis has been but little studied. Bacteriological examination of the sputum usually gives no definite results, since we always find many microörganisms. Many acute cases may be caused by the ordinary pus cocci. At the time of a pneumonia epidemic we have repeatedly seen cases of acute bronchitis with high fever and herpes, but without any signs of pneumonic infiltration. These were probably due to pneumococcus infection. In other cases there is perhaps an influenza infection.

We would add in particular that many acute febrile cases of bronchitis prove on closer investigation (more careful questioning) to be acute exacerbations of a mild chronic bronchitis—"acute recurrent bronchitis." The chronic disease of the bronchial mucous membrane apparently affords a favor-

able opportunity for new infection. In other instances it is merely an acute exacerbation of the old chronic process. Such an acute bronchitis may, of course, occur repeatedly in the same patient.

3. Catarrh of the Finer Bronchi—Capillary Bronchitis.—A simple primary bronchial catarrh rarely extends to the finest bronchi in adults. The secondary bronchitis, however, which develops in other severe diseases (*vide supra*) often extends into the ultimate divisions of the bronchi, and finally leads to the formation of nodules of lobular pneumonia—"catarrhal pneumonia" (*vide infra*). We recognize the implication of the finer bronchi by hearing the high, shrill, whistling rhonchi (sibilant rhonchi), or the abundant fine, moist râles. Respiratory symptoms may be quite marked in extensive catarrh of the finer bronchi. Respiration is evidently accelerated, the inspiration is of the costal type and requires the aid of the accessory respiratory muscles of the neck (the sternomastoids and scaleni), and expiration is usually prolonged. There is often quite a severe cough. The expectoration is mucopurulent and usually not very abundant.

Capillary bronchitis in children is of great practical importance. Bronchitis in young children always has, as experience tells us, a tendency to attack the smaller bronchi. Extensive bronchitis is seen especially in weak children who are rachitic or predisposed to tuberculosis. Children have a great tendency to be attacked with bronchitis at the time of the first dentition.

The parents' attention is usually called to the disease by the appearance of a cough, which is excited especially by the child's crying. Small children never expectorate, for they swallow the secretion which is coughed up into the pharynx. The rapidity of respiration is very striking, it being increased to sixty or eighty, or even more, in a minute. The respiration is also labored, but it is usually superficial, and in severe cases irregular. There is generally a distinct respiratory play of the alæ nasi. We often see a retraction of the lower lateral portions of the thorax on inspiration as a result of the imperfect entrance of air into the smaller bronchi. The expiration is frequently noisy and groaning in children. We hear extensive fine, moist râles over the lungs. In severe cases the child becomes restless, anxious, perhaps markedly pale and cyanotic, and finally apathetic and stupid. In such cases, however, we have no longer to deal with simple bronchitis, but catarrhal pneumonia has already developed. The disease almost always runs its course with fever, the temperature rising to 104° F. (40° C.) and over. The pulse is increased to 120 or 140 or more per minute. The duration of the disease is seldom less than two or three weeks, and it may last much longer. Death may ensue, especially in ill-nourished children, partly as a result of general weakness, and also directly from the imperfect respiration. In such cases we find at the autopsy not only diffuse bronchitis, but also almost always lobular pneumonia. In many cases a gradual recovery finally takes place in spite of the most severe symptoms.

The secondary bronchitis in children complicating measles, whooping cough, diphtheria, etc., has the same tendency to involve the finer bronchi and to lead to lobular pneumonia.

In conclusion, we must mention that acute bronchitis in old persons is also apt to attack the finer bronchi, and it may be dangerous partly from the

general exhaustion, partly from the occurrence of respiratory symptoms and cardiac weakness, as in lobular pneumonia.

Diagnosis.—The diagnosis of bronchitis presents no special difficulty. It is obtained directly by the discovery of rhonchi on auscultation. If these fail, we conclude that there is a mild catarrh of the larger bronchi from the presence of cough and expectoration, if no cause for the cough is to be found in an affection of the larynx. The question is more difficult, but it must always be considered, whether a given bronchitis is a common primary catarrh or secondary to some other affection. This question naturally can be decided only by a very careful examination of the body. We must always remember, furthermore, that severe pulmonary affections may be at first quite latent and show obectively merely the signs of simple bronchitis, while later pneumonia, a tubercular affection, or something similar, develops. A bronchitis which is unilateral, or in which the signs are to be found in circumscribed localities, must therefore be regarded as suspicious. It has long been known that bronchitis in the apices of the lungs, the "apex-catarrh," is often the first objective change to be met with in pulmonary phthisis. We can only conjecture, and not pronounce with certainty on objective evidence, whether nodules of lobular pneumonia are present or not in diffuse bronchitis affecting the finer bronchi.

From what has been said, it is clear that we should be cautious in our prognosis regarding cases of severe bronchitis, especially in children and old people. The prognosis in the milder forms of bronchitis is, of course, always very favorable.

Treatment.—The prophylaxis of primary bronchial catarrh consists in the removal of all the injurious influences mentioned which, as experience shows, may give rise to bronchitis. Careful hardening of the skin to the effects of a change in temperature is of service in persons, particularly children, who have a special tendency to bronchitis, as we have already said in regard to the prophylaxis of laryngitis. It is very important to remember, in this connection, that we can also be successful in our prophylactic measures against secondary bronchitis. Keeping the mouth and pharynx clean, urging deep inspirations, and aiding expectoration by the timely use of tepid baths and shower baths, may often prevent bronchitis or keep it within bounds, when it would surely develop if the patient were neglected.

Simple hygienic measures suffice in the treatment of mild cases of acute bronchitis. The patient should be kept warm, should remain in his room, or, if there be any fever, in bed. Diaphoretic remedies have long been praised as especially potent in the treatment of acute bronchial catarrh. The patient, therefore, should drink hot tea, pectoral tea [1] ("Brustthee"), or elder tea, etc., or hot milk mixed with Seltzer, a remedy whose efficacy is frequently praised by the patient. The tougher the sputum and the more difficult expectoration, the more abundantly should we give warm drinks (Ems water, pectoral tea, etc.). Local treatment of the mucous membrane by inhalations is usually illusory, for only the smallest part of the inhaled fluid reaches the bronchi. We may, however, prescribe inhalations of warm steam, or a one- or two-

[1] A favorite German household remedy, consisting of an infusion of eight parts of althæa, three parts of licorice, one part of orris root, four parts of coltsfoot, and two parts each of mullein and aniseed.—TRANS.

per-cent solution of common salt, especially with a dry cough and a secretion which is hard to loosen.

Otherwise we must employ symptomatic treatment. A mustard plaster or a cold, wet compress about the chest does good service with severe subjective thoracic symptoms. In severe cases a few dry cups may be very useful in adults, but local abstractions of blood are never necessary in simple bronchitis. If there is troublesome cough, so as to disturb the rest at night, we may prescribe small doses of morphin, 5 to 8 grains of Dover's powder (gm. 0.3 to 0.5), 15 to 20 drops of cherry-laurel water, codein, etc. When expectoration is difficult, we may use the so-called expectorants—ipecac, chlorid of ammonium, apomorphin, senega, etc.

We have already repeatedly mentioned the use of tepid baths and shower baths, indicated in severe diffuse bronchitis developing secondarily in the course of other acute diseases.

Warm or tepid baths with somewhat cooler shower baths, two or three times a day, are also to be used as a most powerful remedy in severe cases of capillary bronchitis in children. The baths assist expectoration and guard against the possibility of the development of lobular pneumonia. Wet packs applied to the thorax or over the whole body are serviceable. The child should be wrapped to the neck in a sheet which has been previously dipped in water at a temperature varying with the degree of the fever from 68° to 77° F. (20° to 25° C.) and well wrung out. It is best to leave the arms free. A dry woolen blanket may be wrapped around the moist sheet. This procedure must often be repeated three or four times a day. As to other remedies, we use the same as in adults. With weak children our care must be to keep up the strength by giving the most nourishing food possible and small amounts of wine. An emetic is sometimes indicated in cases with an abundant accumulation of mucus in the bronchi, and is of good service. As experience has shown, we should use opiates for small children only with the greatest caution. Senega and benzoin may be employed as expectorants.

In the bronchitis of old people our chief aim should be to keep up and improve the patient's strength. We prescribe liquor ammonii anisatus, infusion of senega, etc., to aid expectoration, which is usually difficult, since the cough is feeble. We must keep close watch on the condition of the heart (digitalis). Tepid baths may be of advantage, but they must be used with care.

CHAPTER II

CHRONIC BRONCHITIS

(Chronic Bronchial Catarrh)

Ætiology.—Chronic bronchial catarrh may develop gradually from external causes, or in rare cases it may follow acute bronchitis. The same noxious influences which excite acute bronchitis may, by the frequent repetition of their action, result in chronic bronchitis. The constant inhalation of dust is one of the commonest causes of primary chronic bronchitis (see the chapter on Diseases from the Inhalation of Dust). In many cases it is therefore a definite

occupation disease, and is seen in millers, bakers, wool workers, stonecutters, colliers, etc.

In a large number of cases severe chronic bronchial catarrh is not an independent disease, but occurs as a complication or a result of other diseased conditions. The combination of chronic bronchitis with emphysema of the lungs (*vide infra*) is the most common. A large number of cases also are the result of some form of heart disease, such as valvular disease or myocarditis, or of disease of the vessels, leading to stasis in the pulmonary circulation, and finally to a chronic catarrh of the bronchi. Chronic bronchial catarrh in renal diseases also depends in part upon circulatory disturbances, and in part on the toxic action of urinary constituents which have not been normally excreted. Finally, we find a more or less extensive chronic catarrh of the bronchi in other chronic affections of the lungs and pleura, as in tuberculosis or pleurisy.

Chronic bronchitis is seen especially in adults and old people, and more frequently in men, on account of their exposure in the various trades, than in women, but children may also have pronounced cases of chronic diffuse bronchitis. Such cases may often be referred to a previous acute disease of the respiratory organs, especially whooping cough, less often measles, etc. Such cases in childhood often continue into later life, and on careful inquiry we may trace many of the severe cases of chronic bronchitis in adults back to childhood.

Pathological Anatomy.—Chronic bronchitis is characterized anatomically by a persistent and chiefly venous hyperæmia and swelling of the bronchial mucous membrane with increased secretion of mucus and morbid exudation of serum and pus corpuscles. There is often a hyperplasia of the mucous membrane with puffiness of the surface. In old cases, however, we finally meet with an atrophy of all the layers of the mucous membrane. One of the most frequent results of chronic bronchitis is a cylindrical dilatation of the middle and lesser bronchi—bronchiectasis—especially in the lower lobes. This arises gradually from the loss of elasticity of the diseased bronchial walls, and their diminished resistant powers, as well as from the pressure of the stagnating secretions.

Symptoms and Course of the Disease.—The symptoms which are due to chronic bronchitis are dyspnœa, cough, and expectoration. To these should be added the results of a physical examination, in making a diagnosis.

The cough is of very different severity in different cases. Usually it is worse early in the morning, in the evening, and at night, than in the daytime. The amount of expectoration is also subject to great variations. In many cases there is a dry cough (*catarrhe sec, vide infra*), in which only small amounts of tough, viscid sputum are expectorated. In other cases the expectoration is more abundant and mucopurulent, sometimes rather seropurulent, and occasionally thin and separating into layers on standing. In catarrh of the finer bronchi the mucopurulent sputum at times indicates its origin by partly formed casts. We may also note the formation of so-called spirals (*vide infra,* bronchial asthma). Microscopically, the expectoration has no special characteristic appearances, but contains only the usual elements of sputum—pus corpuscles mixed with pavement epithelium, ciliated epithelium, often many bacteria, sometimes needles of fat acids, and rarely a few pointed octa-

hedral crystals, the so-called asthma crystals (*vide infra*). Small amounts of blood may be seen in severe chronic bronchitis, especially in the catarrh of passive congestion, bronchitis with very severe attacks of coughing, etc., but they do not have any bad significance.

Dyspnœa of moderate or even severe degree may be due solely to an extensive catarrh of the finer bronchi with narrowing of their lumen. In many cases in which there is bronchitis the dyspnœa is due chiefly to some coexisting abnormal condition of the heart, the lungs, or the aorta.

Physical Examination.—The percussion in bronchitis shows no special change. At most the resonance may be somewhat tympanitic from the relaxation of the lung tissue, especially in the lower and posterior portions of the lungs, or with an abundant retention of secretion in the bronchi, it may be a little diminished. The inspiratory descent of the lower edge of the lung is lessened when the entrance of air is much impeded by the contracted or plugged bronchi. Auscultation may give either rhonchi, whistling, hissing, humming, etc., or moist râles, according to the extent of the catarrh and the amount and consistence of the secretion. The sounds are usually to be heard over the whole lung, or especially over the lower lobes, because here the catarrh is usually most marked, and retention of secretion is most apt to occur. The respiratory murmur in some places may be quite obscured by the râles. Otherwise it is vesicular, sometimes exaggerated, sometimes rough and indefinite. Expiration is usually prolonged, because the exit of air from the alveoli is much hindered by the narrowed bronchioles. The respiratory murmur may be much diminished, or even entirely suppressed in places where the bronchi are stopped by secretion, which happens most frequently in the lower lobes.

Forms of Chronic Bronchial Catarrh.—Except in mild cases, we usually distinguish several different forms of chronic bronchial catarrh, which may run into one another.

1. Dry Chronic Catarrh.—The dry chronic catarrh (*catarrhe sec* of Laennec) is the form in which the mucous membrane has only a slight secretion. The cough is usually very troublesome and labored, but the patient raises merely a little tough sputum, or none at all. On auscultation we hear sibilant rhonchi, but no moist râles. This form of catarrh is usually associated with pulmonary emphysema, and asthmatic attacks are also frequent. The disease is stubborn, and usually lasts for years.

2. Bronchial Blennorrhea.—In that form of chronic bronchitis termed bronchial blennorrhea there is a very copious seropurulent exudation on the surface of the mucous membrane. The cough is therefore associated with a very abundant and quite thin expectoration, the amount of which in the twenty-four hours may exceed a half pint (quarter of a liter). The expectoration runs together in the sputum-cup and usually separates on standing, the more purulent portion sinking to the bottom, and the seromucous portion, which is usually frothy on the surface, remaining at the top. Numerous moist râles are heard in the lungs, especially in the lower portions. These diminish if large amounts of sputum are coughed up. Anatomically, the bronchi are almost always found dilated in this form of chronic bronchitis.

3. Serous Bronchorrhea.—The so-called serous bronchorrhea (*catarrhe pituiteux* of Laennec) is quite rare but very interesting. It is characterized by the expectoration of a very large amount of frothy, almost purely serous,

or seromucous, thin sputum containing only a slight admixture of pus. The cough usually comes on in very violent paroxysms which last from half an hour to an hour or more. The respiratory symptoms are quite severe, especially during these attacks, and have given rise to the old and useful term "*asthma humidum.*" The expectoration collected in twenty-four hours may amount to one or two quarts (liters). Examination of the lungs usually reveals very abundant and extensive moist râles. The resonance on percussion is normal or a little diminished, from the accumulation of secretion.

The special cause of this peculiar disease is quite obscure. There are mild afebrile and also very severe forms of this type of chronic bronchitis. We have seen several chronic cases, which were attended with persistent fever, and which led to great emaciation and weakness. In one case which came to autopsy there was found a marked tuberculosis of the retrobronchial lymph-glands, while the lungs themselves showed hardly any striking changes. One vagus nerve was wholly imbedded in the tubercular mass of the glands, and it is not impossible that the peculiar attacks of serous expectoration were due to irritation of this nerve. At any rate, we should consider the possibility of such conditions in the future. It is worthy of note that attacks of "humid asthma," with expectoration of large amounts of serous sputum, have also been observed in chronic nephritis, especially in contracted kidney (*q. v.*).

Course of the Disease.—The course of most chronic bronchial catarrhs is very protracted. The disease usually has frequent remissions and fresh exacerbations. The patient is tolerably well in the pleasanter time of the year if he takes good care of himself, but in autumn and winter, or after exposure to various noxious influences, the catarrh grows worse and the patient's symptoms increase. If the disease has lasted for years, we usually find symptoms in the lungs, such as emphysema or chronic tuberculosis, or in the heart, such as secondary dilatation and hypertrophy of the right ventricle, which symptoms gradually become more severe. The details of these conditions are to be found in the appropriate sections.

Diagnosis.—The diagnosis of chronic bronchitis is not difficult in itself, and may easily be made by considering the patient's symptoms and by judging of the result of the physical examination. We must always consider, however, whether the bronchitis is actually a primary disease or a result or a complication of some other chronic disease. Therefore, in every case of chronic bronchitis, the heart and the kidneys (urine) must be carefully examined, as well as the lungs.

In all the severe cases, whenever possible, an X-ray examination should be made. By this means, aneurisms, tumors, and the like have not very infrequently been discovered to be the cause of an existing chronic bronchitis.

Prognosis.—Chronic bronchitis is in most cases a very stubborn affection, which frequently, as we have said, shows improvement, but from which complete recovery is rare. The prognosis also depends greatly upon the patient's circumstances, and upon the possibility of his taking care of himself and avoiding all harmful exposure. In secondary bronchitis the question whether the bronchitis is capable of material improvement or not of course depends mainly upon the prognosis of the primary disease.

The danger in primary chronic bronchitis comes from the final development

of its sequelæ, especially from the gradual appearance of pulmonary emphysema, dilatation of the heart, secondary tuberculosis, etc.

Treatment.—The only hope of success in severe cases in any method of treating chronic bronchitis lies in removing the patient completely, at least for a time, from the action of injurious influences. The favorable result of the baths and health resorts that are employed depends largely upon this, that patients enjoy in them complete bodily rest, and are far better protected from dust and the changes in the weather than at home. We must make the patient comprehend the necessity of this condition as the basis of any treatment. If he cannot go to a suitable climate during the cold season, he must keep his room in all unpleasant weather, but at other times he may be permitted to stay in the open air. Furthermore, the patient must be warned to avoid as completely as possible those harmful influences which his calling and manner of life entail, and among which especially to be mentioned is the bad air in our inns and restaurants. Food should be easily digestible, and, in persons inclined to corpulence, sparingly taken. Alcohol is to be permitted only in a moderate degree. We combat the tendency to constipation, which is often present, by dietetic remedies, by taking fruit, especially grapes, prunes, etc., honey, Graham bread, or by mild laxatives, such as the bitter waters, Friedrichshall, Ofner, etc., since persistent constipation aggravates the patient's symptoms.

If the circumstances of the patient permit and his condition requires it, we should send him south in the autumn in order to avoid the evils of a northern winter, but we must always consider whether the patient has the strength to bear the burden and unavoidable discomfort of such a journey without permanent harm. The rule is to send patients with a bronchial catarrh, when there is much secretion, to health resorts with a dry climate—for example, to the western Riviera, San Remo, Bordighera, Mentone, etc. The somewhat dry yet cooler climate of Meran, Gries, or Arco is suitable for patients with a stronger constitution. Patients with dry bronchitis usually find themselves at their best in a warm but not too dry climate. If we wish to be sure of avoiding the winter's cold, we must choose Sicily, Egypt, or Madeira for a residence. Of the more northern winter resorts we may mention places on the eastern Riviera, especially Nervi.

We must recommend, in bronchitis, a suitable summer residence, particularly for those who dwell in large and dusty cities. Any private country house in a well-wooded and protected place is suitable. If we wish to send patients to a bath, Marienbad, Kissingen, or Homburg are proper places for corpulent people who also suffer from digestive disturbances, while we may send weaker patients to Ems, Soden, Badenweiler, Ischl, Reichenhall, etc. A summer residence on the sea, best on the Baltic, is very serviceable for many patients.

The inhalation treatment is much employed in chronic bronchitis, but we should not cherish too high hopes about its use. The best inhalations in dry catarrhs are simple steam, a one- or two-per-cent solution of common salt or bicarbonate of soda, Ems water, etc. In cases with marked secretion, inhalations of oil of turpentine are most to be praised. The simplest way is to pour a teaspoonful of oil of turpentine into hot water and inhale the vapor as it arises. The so-called turpentine pipe, however, is more convenient and more efficacious (Fig. 39). This consists of a flask, which is filled to the height

of several inches with water and then with a layer of oil of turpentine or of *oleum pini pumilionis* (P. G.)., some 2 cm. thick. Two glass tubes, open at both ends, are passed through the cork. One straight tube extends down into the layer of water: the lower end of the other is free in the upper part of the flask. The outer portion of this last tube is bent at an angle and is connected with the mouthpiece through which the patient inhales. The formation of turpentine vapor is aided by putting the flask in hot water. We have treated many patients in this way, who, for a number of hours a day, "smoked" their turpentine pipes.

Fig. 39.—Turpentine pipe.

In treating chronic bronchitis the pneumatic method was considerably employed for a time; that is, the patient was made to breathe artificially compressed air, or to expire into air of less than the atmospheric pressure, by means of a movable pneumatic apparatus, as proposed by Waldenburg and others. Of late, however, this method of treatment has received less favor, inasmuch as actual results have fallen decidedly short of the benefit promised. In Ems, Reichenhall, and other places, special pneumatic cabinets have been arranged, filled with compressed air, in which patients remain for varying lengths of time.

I consider the exhibition of methodically given sweat baths, and, above all, the electric-incandescent-light baths (the so-called Kellogg [1] incandescent-light baths) far more effectual than the pneumatic treatment. In these baths the patient is comfortably seated in the cabinet, his arms are free, and his head is outside the cabinet and covered with an ice cap or cool cloths. The severe cases of bronchitis require especial care; while the patients are in bed they may be given localized electric-light treatment to the trunk and chest by means of specially devised contrivances. I have seen very satisfactory results from this method of treatment, even in advanced cases. The light baths are given daily for about fifteen to twenty minutes until there is profuse sweating all over the body. The patients are next placed in a warm bath, are then rubbed dry, and remain quiet for one to two hours. If an electric incandescent light bath is not at one's disposal, the usual steam cabinets and other diaphoretic measures may be employed. All these methods of treatment, of course, require careful supervision of the patient by the physician.

One of the most serviceable remedies in dry chronic bronchitis is a large amount of warm drink (pectoral tea, Ems water, Seltzer, etc.). Iodin preparations (sodium iodid, potassium iodid, sajodin, etc.) sometimes act favorably in making a tough secretion fluid. Of expectorants, ipecac and apomorphin are most recommended in this form of bronchitis. For distressing cough we may give Dover's powder, morphin, or codein. In bronchial blennorrhea

[1] [Devised by J. H. Kellogg, of Battle Creek, Mich.]

we know empirically that the internal use of balsams causes a distinct diminution of the secretion. Oil of turpentine is the most active, and may be given internally in gelatin capsules, or mixed with milk, in doses of 10 or 15 drops three or four times a day. After each dose of turpentine we should give a glass of milk. Lépine, G. Sée, and other French physicians recommend terpin hydrate as still more effectual. This is a derivative of turpentine, and is best employed in pills containing 1.5 gr. (gm. 0.1), of which two or even more are to be taken three times a day. It may also be given in solution as follows: Terpin hydrate, ʒijss. (gm. 10); alcohol, q. s. ad sol. faciendam; aq. dest., ℥vij (gm. 200). Misce. Sig.: Two or three spoonfuls daily. Myrtol [terebene], balsam of copaiba, and balsam of Peru are also used internally. The chief expectorants are infusion of senega root, liquor ammonii anisatus, etc. We should be very sparing of narcotics at first, but in severe cases we cannot wholly dispense with them.

[The iodid of potassium in doses of 5 to 10 gr. thrice daily is sometimes distinctly curative. An outdoor life, free diet, moderate alcoholic stimulus, tonics, and woolen clothing do much to promote recovery.]

Local applications to the chest in the form of embrocations, mustard plasters, dry cups, or cool or warm wet compresses are to be used, especially with severe dyspnœa, or with pain and a feeling of oppression in the chest.

In all secondary chronic catarrhs our chief attention, beyond the symptomatic treatment of the bronchitis, must be directed to the treatment of the underlying disease. If we succeed in once more regulating the heart's action by digitalis when there is uncompensated heart disease, or in establishing diuresis when there is renal disease, or in improving the general physical condition by proper dietetic measures in the corpulent, gouty, alcoholic, etc., we may also in that way cause improvement in the existing bronchial catarrh.

CHAPTER III

FETID BRONCHITIS

(Putrid Bronchitis)

Ætiology.—By putrid or fetid bronchitis we mean that form of bronchitis in which the secretion of the mucous membrane undergoes a putrid decomposition, and in which, consequently, the expectoration takes on a peculiar and extremely foul odor. The special agents which cause fetid bronchitis are still unknown.

The opportunity for the agents of putrefaction to enter the bronchi with the inspired air is, of course, often given, but a fetid bronchitis naturally is excited only when they can remain there and increase. Fetid bronchitis therefore develops quite rarely in a previously healthy lung (primary fetid bronchitis). The retention and the further development of the bacteria of putrefaction are chiefly favored, as we know, by diseased conditions which already exist in the bronchi. A great number of cases of fetid bronchial catarrh therefore develop secondarily upon other pulmonary affections of longer standing. Thus the expectoration may quite suddenly change and take on a fetid char-

acter in the course of a chronic or rarely of an acute bronchitis or of phthisis. Bronchiectasis (*vide infra*) greatly favors the development of this putrid change, for in it the retention and stagnation of large amounts of secretion promote and occasion the putrid decomposition. If a putrid decomposition of the secretion begins in one part of the bronchial system, the further extension of the process follows from direct infection.

In rare cases putrid bronchitis also develops as a result of an embolic pulmonary gangrene.

Symptoms and Course; Anatomical Changes.—If a fetid bronchitis arises in the course of some other chronic pulmonary disease, its appearance may be marked by a sudden impairment of the general condition, by high fever, often associated with numerous chills, and by an increase of the thoracic symptoms, such as pain and cough. The change in the expectoration, the peculiarity of which was first accurately described by Traube, is characteristic. The expectoration shows the same character in cases of apparently primary fetid bronchitis.

The sputum has a very repulsive, sweetish, putrid smell. The expectoration is usually quite abundant; the consistence is rather thin. On standing, the sputum shows a very marked division into three layers. This division is due to the abundant serous exudation in the bronchial mucous membrane and to the thin fluid character of the expectoration, which permits an unequal division and a settling of the solid constituents. The upper layer consists of a very frothy, mucopurulent stratum, consisting in part of individual masses, from which a number of coarser or finer fibers float down into the middle layer. This middle layer consists of a dirty-green mucoserous fluid. At the bottom of the vessel is found the third layer, which is often the thickest, and is composed entirely of pus. It consists of pus corpuscles which have sunk to the bottom, and is of a rather thin, greasy consistence. With the naked eye we generally recognize a number of little whitish-gray plugs and particles in it. These so-called "Dittrich's plugs," which are easily crushed under a cover-glass, are quite characteristic. Microscopically, they consist of decomposed pus corpuscles, detritus, and bacteria, and usually contain very many needles of fat acids arranged in bundles (see Fig. 40). We often find also in the sputum large masses of fungi, especially great bunches of twisted leptothrix fibers, which, by an unpracticed eye, may readily be mistaken for elastic fibers. The latter are, of course, never found in the expectoration of a simple fetid bronchitis, but only in the deep-seated, destructive processes in the lung, like gangrene. On chemical examination of the sputum, the ordinary products of putrefaction may be found—volatile fat acids, especially butyric and valerianic acids, also sulphureted hydrogen, leucin, tyrosin, etc.

FIG. 40.—Crystals of fatty acids.

It is very characteristic of many cases of fetid bronchitis that the sputum does not have a putrid character at all times. Sometimes for days the patient coughs up a simple catarrhal secretion, and then all at once comes an outburst of intolerable stinking expectoration. This is probably because a circum-

scribed putrid focus is frequently shut off, the expectoration coming solely from the other bronchi, where there is merely a simple catarrh, until finally the accumulation of putrid secretion is all at once coughed up again in large amount by mouthfuls.

When fetid sputum is coughed up, the patient's breath also becomes very foul-smelling, so that he often becomes a burden to his associates.

The signs which fetid bronchitis gives on physical examination are those of an ordinary bronchitis; but it is characteristic of many cases, in distinction from ordinary chronic bronchitis, that the physical signs (râles, dry rhonchi) are limited chiefly to a definite portion of the lung, usually one lower lobe. In a great number of cases we also find signs of consolidation and contraction of the lung, of pleurisy, etc., which do not belong to fetid bronchitis as such, but are due to complications or sequelæ.

The most frequent of these sequelæ is the development of a "reactive" lobular inflammation, a genuine pneumonia which follows a catarrh of the finer bronchi. These reactive inflammations, in part exaggeration of the existing bronchitis, in part lobular pneumonias, frequently occur in separate attacks. Quite suddenly, increased cough and expectoration appear, accompanied by darting pains in the side, fever, etc. After several days—sometimes, however, only after two to three weeks or even longer—these manifestations disappear, to be followed by an afebrile period with milder symptoms. In the lobe of the lung which is the main seat of the fetid bronchitis, a chronic interstitial pneumonia, associated with more or less retraction, pleuritic adhesions, etc., gradually develops. Special mention must be made of the danger of the development of gangrene. The putrefying agents having once gained entrance into the lungs, any subsequent pneumonia may be followed by pulmonary gangrene. For this reason we not infrequently find, post-mortem, isolated smaller or larger gangrenous foci in conjunction with a fetid bronchitis. In such cases the fetid bronchitis is certainly the primary factor, and the development of gangrenous foci the secondary lesion. It will be shown later that the reversed condition may occur. Fetid bronchitis and gangrene of the lungs run into each other so often, both clinically and anatomically, that there is no sharp line to be drawn between them. If the nodules are superficial, and reach the pleura, the infection attacks this, and we have a purulent or even an ichorous pleurisy.

The smaller and medium-sized bronchi are almost always found in a condition of cylindrical dilatation in long-standing fetid bronchitis. Their mucous membrane is intensely inflamed, and often ulcerated superficially. On its surface we see in the cadaver the greasy purulent masses and the plugs which we find in the expectoration during life.

Whatever may be the case with the general course of fetid bronchitis, its beginning is often quite sudden and acute, both in the primary and in the secondary forms, as we have said. The patient is attacked with fever, which may often be quite high, and with a stitch in the side, cough, and expectoration. Later, the characteristic peculiarities described above appear. The further course of the disease is almost always chronic, lasting for years, but subject to many variations. Very often manifest improvement, and even apparent recovery, takes place, when suddenly there is a new attack of fever and thoracic symptoms. The general condition and nutrition of the patient may be quite

good for a long time, except during the periods of marked exacerbation of the disease. Patients with chronic fetid bronchitis often appear somewhat bloated, but also pale and slightly cyanotic. Peculiar clubbed thickenings of the terminal phalanges of the fingers, or more rarely of the toes, gradually develop, as in many cases of bronchiectasis. Slight œdema of the lower extremities is also sometimes present.

Symptoms referable to other organs may be wholly absent. We see most frequently disturbances of the stomach, loss of appetite, and nausea, which probably come from swallowing the fetid sputum. Patients also complain of occasional rheumatic pains in the muscles and joints, which are probably due to an absorption of septic matter. In conclusion it must be mentioned that, in fortunately rare cases of fetid pulmonary disease, pyogenic germs reach the brain by metastasis, and give rise to purulent meningitis or cerebral abscess.

The danger of the disease, apart from the exceptional occurrence just mentioned, lies in the possible extension of the process to the lungs and the development of pulmonary gangrene and its sequelæ. We hardly ever find a simple fetid bronchitis in the cadaver, but we almost always see other processes besides, which have been mentioned above—reactive pneumonia, pulmonary gangrene, pleurisy, etc. All these processes develop very readily, and make rapid progress in old, decrepit persons, who live under bad hygienic conditions, in whom putrid processes in the lungs are frequent.

Diagnosis.—The diagnosis of fetid bronchitis is not difficult in itself, for the diagnosis of a putrid process in the lung may be made from the stinking sputum alone. It may be difficult to decide whether we have to do merely with a fetid bronchitis, or with a pulmonary gangrene also. Decisive indications of pulmonary gangrene are derived from physical examination—dullness, bronchial respiration, and coarse, moist râles, signs of a cavity—and also the discovery of elastic fibers and fragments of parenchyma in the expectoration.

Prognosis.—The prognosis must be made with care in every case of fetid bronchitis. If the external circumstances of the patient are favorable, he may remain in tolerable health for years. We must always be prepared for the appearance of new exacerbations of the disease and of affections of the lung itself, and must also bear in mind the rarer sequelæ (empyema, abscess of the brain). Complete cure is exceptional.

Treatment.—The chief aim of treatment must be to bring the putrid processes in the bronchi to a standstill by the death of the agents of putrefaction. The difficulty of fulfilling this task lies in the impossibility of getting the disinfecting material to act on the bronchial mucous membrane in the necessary amount and concentration. Nevertheless, we can, without doubt, at least relieve a fetid bronchitis and keep it in check by the judicious use of inhalations. Inhalations of a two-per-cent solution of carbolic acid are most useful, given for five or ten minutes several times a day. These, however, are sometimes not well borne if long continued, and they may excite mild symptoms of carbolic poisoning—such as headache, malaise, and dark carbolic urine. We have often used with good results the "carbolic mask" recommended by Curschmann, a kind of respirator fastened in front of the nose and mouth, containing cotton in a special receptacle impregnated with carbolic acid, equal parts of carbolic acid and alcohol, or other remedies such as turpentine or creosote. Many patients can wear these masks, with occasional interruptions, for many

hours a day. Besides carbolic acid we may use inhalations of turpentine (turpentine pipe or vapor), oleum pini pumilionis, creosote, etc. The internal use of these and similar remedies also seems frequently to be of service in putrid bronchitis. We recommend especially the internal use of turpentine (in gelatin capsules), myrtol (also in gelatin capsules), also terpin hydrate (six to ten 2-gr. [gm. 0.1] pills a day), creosote, syrupus kalii sulphoguaiacolici (sirolin), etc.

In other respects all the general hygienic and symptomatic methods of treatment recommended for common chronic bronchitis (expectorants, narcotics, fresh-air rest cures, climatic health resorts, etc.) are also useful in fetid bronchitis. The sputum should be disinfected by putting strong carbolic acid, etc., into the sputum-cup to lessen the bad odor. It is a very good plan to keep the carbolic spray at work in the patient's room as often and as long as possible; or the air may be impregnated with oleum pini pumilionis.

CHAPTER IV

CROUPOUS BRONCHITIS

(Fibrinous or Pseudo-membranous Bronchitis)

Croupous bronchitis is a peculiar form of disease of the bronchial mucous membrane, of very rare occurrence, in which there is a formation of extensive fibrinous patches in the bronchi. Only that form of croupous bronchitis which occurs primarily in the bronchi is to be considered here, and not the secondary form, which on the one side is associated with diphtheria in the pharynx and larynx, and on the other with croupous pneumonia.

Ætiology.—The ætiology of the disease is as yet wholly unknown. From analogy with other well-known croupous inflammations of mucous membranes, we must look here for some noxious influence which destroys the epithelium, but up to this time we are entirely ignorant of its character. Individuals in youth and middle age, somewhere between ten and thirty years old, are the chief victims. Men are attacked somewhat more frequently than women. The disease comes on either in persons who were previously healthy—the essential fibrinous or croupous bronchitis—or in those who have already suffered from some other disease, especially some chronic pulmonary affection—the symptomatic, secondary croupous bronchitis. It is not certain whether the last-named cases have the same ætiological relations as the cases of genuine primary fibrinous bronchitis. Fibrinous bronchitis has been observed in the course of typhoid fever and other acute infectious diseases.

Symptoms and Course.—Primary fibrinous bronchitis occurs in two forms, acute and chronic. The acute form begins quite suddenly, with fever, cough, pain in the chest, and, as a rule, severe dyspnœa. The fibrinous coagula, which alone render the diagnosis possible, appear in the expectoration either at once, or after the existence for some days of what is apparently simple catarrhal bronchitis.

These coagula form complete casts of the bronchi, and are more or less branching. They are of a whitish color and of quite a dense, elastic con-

sistence. The main stem may be a centimeter thick, and from it the further ramifications extend, dividing dichotomously. The largest casts are 10 or 15 cm. long. On section, we usually find a free lumen within, and generally recognize a definite laminated structure in the membrane. In many places they are enlarged and swollen. Microscopically, we find white blood corpuscles in and upon the hyaline ground substance of the casts, and also red blood corpuscles, sometimes epithelial cells, and quite often the peculiar pointed octahedral crystals which are also found in the expectoration in bronchial asthma (*vide infra*). The so-called "spirals" (*vide infra*) have also been found in the expectoration of fibrinous bronchitis. Chemically the casts apparently consist of coagulated albumen. It is doubtful whether they actually consist of fibrin. Weigert's fibrin stain does not affect them. Their solubility in alkalies, especially in lime water, is of therapeutic importance.

On coughing, the patient usually raises a simple mucous or mucopurulent expectoration besides the casts, and in this sputum the casts are imbedded. They are often first discovered by pouring the whole amount of sputum into water, when they unfold and spread out. The expectoration also contains not infrequently a slight admixture of blood.

The subjective symptoms of the patient may be very violent. The dyspnœa sometimes attains a high and alarming degree. It ceases when a large cast is expectorated after a severe paroxysm of coughing. Such attacks may recur every day or two. In other cases, however, the subjective symptoms are comparatively slight.

Physical examination of the lungs reveals little that is characteristic. In uncomplicated cases percussion shows nothing abnormal, or at most the signs of an acute emphysema. Auscultation gives the ordinary signs of bronchitis, not characteristic in themselves, such as rhonchi or moist râles. If a large bronchus is plugged, the respiratory excursions and the respiratory murmur are almost entirely absent in the corresponding portion of the lung, but after the expectoration of a cast respiration becomes once more audible.

The duration of acute cases is sometimes only a few days, at most a few weeks. In favorable cases the fever, which at times is quite high, soon disappears, the respiratory symptoms grow milder, the expectoration of the casts ceases, and there is a complete and permanent recovery. In severe cases, however, death often ensues with all the symptoms of suffocation. The acute form sometimes becomes chronic, but this is rare.

The chronic form of fibrinous bronchitis may last for years. Usually the condition grows worse periodically, at varying intervals of time, and at each exacerbation casts are expectorated, while in the interval there is apparently merely a simple bronchial catarrh. Some observations are also recorded in medical literature of persons who have expectorated these casts at intervals for years without any special disturbance of their health or their nutrition. In some cases other chronic pulmonary affections, such as tuberculosis, finally develop.

The pathological anatomy of fibrinous bronchitis is not yet satisfactorily known on account of the rarity of the affection. The changes in the lungs found at the autopsy of fatal cases have usually been complications, such as pneumonia, pleurisy, or tuberculosis, which stood in no direct relation to the fibrinous bronchitis.

Prognosis.—In all acute cases the prognosis should be guarded, for we know that about one fourth of the cases terminate fatally. The chronic cases, as has been said, are usually very protracted and are subject to frequent exacerbations, but they differ from the acute cases in being much less dangerous.

Treatment.—We make special use for inhalations of those remedies which, as we have said, have power to dissolve the casts. We usually employ a two- to five-per-cent solution of carbonate or bicarbonate of sodium, and above all lime water, either pure or diluted with an equal volume of water. The internal administration of iodid of potassium, in doses of 20 to 45 gr. (gm. 1.5 to 3.0) a day, proves of advantage in many cases. Energetic inunction with mercurial ointment is sometimes of service. In one tedious case I tried a course of sweating without permanent benefit. Expectoration of the casts may be aided in many cases by such drugs as senega and benzoic acid, or by the timely use of emetics. We do not know any remedies which can prevent a return of the attacks in the chronic form. The treatment, except at the time of the attacks, is the same as in ordinary chronic bronchial catarrh.

CHAPTER V

WHOOPING COUGH

(Pertussis. Tussis convulsiva)

Ætiology.—By the name "whooping cough" we mean a specific disease of the mucous membrane of the air-passages, which is seen chiefly in children, and is characterized by a peculiar violent and paroxysmal cough. Sporadic cases are of almost constant occurrence in large cities, but the disease often appears in epidemic outbreaks. Epidemics of whooping cough follow epidemics of measles with remarkable frequency.

Whooping cough is without doubt contagious, and therefore often attacks one child after another in the same family. Kindergartens, orphan asylums, and day nurseries aid very much in extending the disease. The contagious element seems to be connected with the air expired by the patient, particularly with the mucous secretion coughed up. Children are most subject to an attack up to the age of six years; from that age the liability to the disease decreases rapidly with increasing years. Whooping cough is seen, indeed, in adults, but it is quite rare, and almost always without the pronounced features of tussis convulsiva.

The epidemic onset, the contagiousness, and the whole course of the disease favor the theory of its infectious nature. The presence of the organisms which are supposed to be the poison of the disease has not yet been certainly demonstrated, although many have claimed to discover characteristic bacteria in the sputa of patients. In an epidemic in Hamburg, Jochmann almost constantly found a bacillus present that closely resembled that of influenza. However, the question as to the cause of whooping cough is not definitely solved. If a patient has once had the disease, he is almost invariably safe from a second attack.

Symptoms and Course of the Disease.—Whooping cough begins with the symptoms of a catarrh of the trachea and bronchi, which develops more or less rapidly, and which at first often shows nothing characteristic. We can at this period make a tolerably probable diagnosis only at a time when an epidemic is prevailing, or in case the child's associates have already been attacked with the disease. The cough is often very persistent, obstinate, and severe at the beginning, but it does not yet come on in distinct paroxysms. Examination of the chest shows nothing peculiar except a few rhonchi. There is often a coryza, with frequent sneezing, and there is sometimes a mild conjunctivitis. The child is restless and feverish, especialy toward night. The temperature may repeatedly reach 103° or 104° F. (39° to 40° C.) in this initial fever. The duration of this first so-called catarrhal stage varies, but it usually lasts a week or ten days.

The catarrhal stage gradually passes into the second, convulsive stage, without any sharp boundary. The cough becomes more violent, and comes on in the separate paroxysms of whooping cough which are characteristic of the disease. We do not know the particular reason why the cough has this paroxysmal character, but a nervous factor probably plays the chief part in it.

The peculiarity of the attack consists in the violent, paroxysmal fits of coughing, which are from time to time interrupted by deep, long-drawn, loud, and shrill inspirations, due to the occurrence of a spasmodic contraction of the glottis. Exceptionally there are cases without this loud whistling inspiration. The child becomes markedly cyanotic during the attack, the veins in the neck swell, and tears come into the eyes. Hemorrhage into the conjunctiva, nosebleed, and in some cases hemorrhages into other organs, as the ear and the skin, often come on as a result of this stasis. Vomiting very often occurs either during a paroxysm or at its close. Involuntary evacuations of urine and feces may also follow from the violent contraction of the abdominal muscles. Exceptionally we observe still more severe symptoms with a paroxysm: a complete spasmodic cessation of respiration with imminent danger of suffocation, or sometimes general convulsions. In two cases we have seen hemiplegia in children which, according to the positive statements of their parents, came on suddenly during a severe attack of whooping cough. It is still undetermined whether these "whooping-cough hemiplegias" are due to an actual cerebral hemorrhage or whether they come from the enormous venous stasis in the brain.

The paroxysms vary with the severity of the disease, frequently appearing ten or fifteen times in twenty-four hours; sometimes with greater frequency—fifty times or more. They also occur at night as often or even oftener than in the daytime. They come on either spontaneously or from some special predisposing cause. The attacks which come on during eating cause the most disturbance, because the food taken is almost always vomited again. In children with whooping cough we can often excite a spasm at any time—and this is important in diagnosis—by putting a spatula in the mouth, by pressing on the larynx, or by making the child cry. If there are several children with whooping cough in the same room and a paroxysm attacks one of them, the others, as a rule, soon begin to cough, too. Some prodromal symptoms often precede the peculiar paroxysm, such as general uneasiness, rapid respiration, or vomiting. At the end of a paroxysm many children are very feeble and

exhausted, but others recover rapidly, and are playing again quite briskly a few minutes after.

In general the child feels quite well in the interval between the paroxysms, but the effects of the violent attacks of coughing may, of course, often be seen. Besides the occasional hemorrhages into the conjunctiva, we find the eyelids somewhat swollen, their veins dilated and blue, and visible through the skin. Many children grow very thin from the repeated vomiting. A small ulcer is quite frequently formed on the frænum of the tongue, the origin of which is to be referred to mechanical causes. The tongue is violently protruded in the severe paroxysms of coughing, and the frænum is thus torn, or injured by the sharp lower incisors.

Physical examination of the lungs shows nothing abnormal in uncomplicated cases except a few moist râles or rhonchi. Sometimes the rhonchi are wanting, or are present in small numbers only a short time before a paroxysm, but in other cases an intense diffuse bronchitis is developed, which often leads to the development of a lobular pneumonia (*vide infra*). Sometimes, but not always, there is an acute catarrhal inflammation of the bronchi, and especially of the posterior wall of the larynx.

The fever, which is usually present in the first or catarrhal stage, is absent in the convulsive stage. The child is free from fever for the most part. We often find a slight rise of temperature up to 100° or 101° F. (38° to 38.5° C.), but only toward night. Higher and more persistent fever points to the development of complications, especially on the part of the lungs.

The convulsive stage seldom lasts less than three or four weeks, and often much longer, up to three or four months. The paroxysms gradually become less frequent and less violent (*stadium decrementi*), until they finally disappear entirely; but relapses and fresh exacerbations also occur in this stage. A certain "irritability" of the bronchial mucous membrane remains for a long time after whooping cough. Finally, however, the disease, in uncomplicated cases, goes on to a permanent and complete recovery.

Complications and Sequelæ.—The severe results which sometimes follow whooping cough are probably due in part to the direct action of the specific causes of the disease, and in part to complications of a secondary nature whose development is merely favored by the whooping cough. The most important are complications in the lungs. A lobular catarrhal pneumonia often develops after a severe bronchitis which involves the finer bronchi. In such cases the respiration becomes hurried and superficial, the fever higher, and the general condition bad even in the times between the paroxysms. On examination of the lungs we hear numerous moist râles, especially over the lower lobes; and we can sometimes make out dullness on one or both sides, if there is extensive pneumonic infiltration. Such cases are always very protracted, and many children succumb, partly from the disturbance of respiration and partly from general weakness and inanition.

Complications in other organs are much rarer. Among the most frequent are attacks of diarrhea which impair the child's nutrition. Many observers have also mentioned the quite frequent occurrence of a croupous or diphtheritic inflammation in the pharynx and larynx in the course of whooping cough. Finally, a case under our own observation may here be mentioned, in which death occurred with severe nervous symptoms, convulsions, and coma.

At the autopsy very numerous capillary hemorrhages were found in the brain.

Pulmonary emphysema is the first thing to be mentioned among the sequelæ of whooping cough. From the marked pressure which the severe and frequent outbursts of coughing exert from within upon the alveoli of the lungs, they gradually become dilated. An acute lobular emphysema ("acute pulmonary inflation") is set up, which sometimes passes into a typical chronic pulmonary emphysema (*q. v.*). Chronic bronchial catarrh may also remain for a long time after an attack of whooping cough. We have already stated (see page 197) that many incurable cases of chronic bronchitis in adults are to be referred to an attack of whooping cough in childhood.

A third important sequel of whooping cough is pulmonary tuberculosis. The bronchitis and lobular pneumonia which occur during whooping cough sometimes do not improve, especially in weak children with a tuberculous tendency. The fever continues high, the child grows thin, and constantly becomes more and more miserable. At the autopsy we find cheesy nodules in the lungs, cheesy bronchial glands, and at times tuberculosis of other organs. These cases signify that when a tuberculous infection is present, but is still latent, the whooping cough acts as an exciting cause for the outbreak of the disease, or that a greater receptivity to infection with tuberculous poison is created by the whooping cough. Möbius has lately reported the occurrence, in a few cases, of paralysis as a sequel of whooping cough. This usually begins in the lower and extends to the upper extremities, and is due apparently to neuritis.

Diagnosis.—The diagnosis of whooping cough cannot be made with certainty, as we have said, until the second or convulsive stage. It is easy then, however, since the characteristic attacks occur in no other affection of the lungs in like manner and with like frequency and duration. If we have no opportunity to observe the attack itself, and have to depend upon the description of the friends, the diagnosis is sometimes more uncertain; but the accounts of the occurrence of cough, in individual paroxysms associated with vomiting, are usually so characteristic that errors are, on the whole, rare. Furthermore, the child between the paroxysms usually presents certain signs: he has a bloated aspect, or we may find slight hemorrhages into the conjunctiva, or ulcers on the frænum of the tongue, which make the diagnosis highly probable. Under some circumstances we may also make the attempt to bring on the paroxysm artificially (*vide supra*). In adults, as we have said, the attacks are rarely as characteristic as in children. There are usually the symptoms of a more or less severe bronchitis with obstinate paroxysmal cough, but without the characteristic attacks and usually without vomiting. The diagnosis of whooping cough, therefore, rests mainly upon the existence of special ætiological conditions (the coexistence of the disease in children, etc.).

Prognosis.—The prognosis is favorable with the majority of children if they are previously strong and healthy. Very young children are in more danger than older ones. [Under two years there is great danger, and over five scarcely any. It has been said that whooping cough causes one fourth the total mortality of children in London.] There is danger if secondary pneumonia develops, and if the general nutrition and strength of the child suffer. As soon as the diagnosis is certain we must call the attention of the parents

to the probable long duration of the disease. Regard must also be paid to the possibility of the development of sequelæ, especially in weak children suspected of tuberculosis. I am convinced that many cases of lifelong chronic bronchitis are referable in the final analysis to an attack of whooping cough in childhood.

Treatment.—Since the disease is protracted and is not devoid of danger, it is our duty, when an epidemic of whooping cough prevails, to guard children from it as far as possible. If one child in a family is taken ill, the other children must be rigorously kept away from him. If circumstances permit, it is better to send them away to another place free from whooping cough.

With regard to the treatment of the disease, we must first endeavor to fulfill general dietetic and hygienic indications. The child should breathe good, pure air, and for this reason it is often advisable to transfer the patient to a larger room, with as much air and sunlight as possible. The atmosphere should not be too dry, and it is advisable to employ a spray of water (carbolic solution) frequently, or to hang up moist sheets in the room. In good weather the child should be out of doors a large part of the time, provided fever has ceased. City children are to be sent, if possible, into the country. The food should be good and nourishing, but dry and crumbly articles should be avoided, being apt to excite cough. Warm or lukewarm baths frequently prove very beneficial, particularly when there is considerable bronchitis, as they lessen the danger of a lobular pneumonia.

The medicinal treatment of whooping cough has not yet shown brilliant results despite the large number of remedies recommended. During the catarrhal stage it is usually sufficient to give a simple expectorant (ipecac, etc.) and plenty of warm drink. In the convulsive stage the best remedies to try are quinin, antipyrin, belladonna, bromid of potassium, and bromoform. The latter has been much recommended of late. Quinin is given in powders of 1.5 to 8 gr. (gm. 0.1 to 0.5) several times a day, either in capsules, or, in the case of smaller children, with chocolate. The earlier this remedy is employed the more prompt is said to be its beneficial influence. Antipyrin is at present used more frequently than quinin, and in doses of 4 to 8 gr. (gm. 0.25 to 0.5) several times a day it often produces distinct improvement. Belladonna is prescribed in powders containing $\frac{1}{12}$ to $\frac{1}{6}$ gr. (gm. 0.005 to 0.01) of the extract of belladonna, giving three to five such powders a day. This remedy has often seemed to the author to diminish the number and violence of the paroxysms. Bromid of potassium is employed in an aqueous solution in the dose of 15 to 45 gr. *per diem* (gm. 1 to 3). Its efficacy is probably due to its power to diminish reflex excitability. The same drug employed in an atomizer often has a palliative effect. Bromoform is the remedy most used of late; two to five drops or more are given several times a day in sweetened water or fruit juice. It is readily taken, and it seems to act favorably both on the severity of the individual attacks and on the whole course of the disease. Finally, we might mention a recent preparation called "pertussin" that has been widely praised for its results. It is a fluid extract of thyme, and is given in teaspoonful doses three or four times a day. Pyrenol (gr. v [gm. 0.3] three times a day) is also said to be effectual.

If the paroxysms are very violent we may cautiously administer small doses of morphin or codeia. Inhalations of chloroform and ether have also been recommended. The following mixture is a suitable one:

℞ Chloroformi ..... ℥j (30 c.c.);
Ætheris ..... ℥ij (60 c.c.);
Ol. terebinthinæ ..... ℥ijss. (10 c.c.).
M.

Sig. One or two teaspoonfuls to be poured upon a pocket handkerchief for inhalation.

Finally, efforts have been repeatedly made to lessen the frequency and the severity of the attacks by anæsthetizing the pharynx and larynx by painting with a ten- to fifteen-per-cent solution of cocain. Michael advocates the daily insufflation into the nostrils of powdered benzoin. Neither method of treatment has succeeded in establishing itself in practice. Numerous attempts have been made to reduce the number of paroxysms by inhalations of the most diverse remedies. Because of the infectious nature of the disease, frequent inhalations of one- to two-per-cent solutions of carbolic acid were employed. The results were not striking, and the danger of poisoning (as evidenced by the urine) must not be lost sight of. Turpentine or benzine, 20 to 30 drops poured on a sponge moistened with hot water, is more to be recommended. Inhalations of bromid and salt solution (sodii bromidi, sodii chloridi, āā gr. xxxviij [gm. 2.5], aq. destillat., ℥vij [gm. 200]) are also worthy of trial. Turpentine and especially terpin hydrate (gr. vij, gm. 0.5, thrice daily) are also given internally. [Farlow and others report marked success from spraying the upper air-passages with a two-per-cent solution of resorcin.]

In many cases we finally content ourselves with taking good general care of the child, and, if the weather permit, with keeping him as much as possible in the open air. For the detailed treatment of the complications and sequelæ of whooping cough the reader is referred to the appropriate chapters of this book.

CHAPTER VI

BRONCHIECTASIS

(Bronchial Dilatation)

Dilatation of the bronchi is usually not a separate disease, but it is a result of various affections of the lungs and bronchi. Nevertheless, we will speak of it briefly in this connection since many cases actually present a very characteristic clinical picture.

We distinguish anatomically the cylindrical and saccular bronchiectases.

CYLINDRICAL BRONCHIECTASIS

Cylindrical bronchiectasis consists of a uniform dilatation of a bronchial tube, and occurs most frequently in the medium-sized, or rarely in the finer

bronchi of one or more lobes of the lung. We usually find at the autopsy that, on slitting up the bronchi, the point of the scissors can easily be pushed through the dilated bronchial tube close up to the pleura. Cylindrical bronchiectasis is usually due to a long-continued bronchitis, and develops most frequently in cases of emphysema, and also in whooping cough, measles, and sometimes in phthisis, etc. The primary process is probably always the atrophy which follows the catarrh, and the diminished resistance of the bronchial walls thus occasioned. The dilatation of the lumen of the bronchus is produced gradually, partly by the traction of the thorax during inspiration, and still more by the increased pressure in the bronchi due to the frequent and violent fits of coughing, and finally, perhaps, by the constant pressure of the stagnating secretion.

The diagnosis of cylindrical dilatation of the bronchi is only a probable one. We suspect that a bronchiectasis has formed if the conditions are fulfilled which we know lead to it. In the chronic bronchial catarrh of emphysema we judge that there is cylindrical dilatation of the bronchi if the secretion is very abundant and comparatively thin, and separates into layers on standing in a sputum-cup. The dilatation is usually emptied by a severe paroxysm of coughing, such as is apt to occur in the morning if the secretion collects in great quantity during the night. Physical examination usually gives numerous, feeble, fine, and medium nonresonant moist râles, especially in the lower lobes. The respiratory murmur sometimes loses its vesicular character in marked cylindrical bronchiectasis, and has a more indefinite and blowing or whispering quality. Sometimes it is quite obscured in the lower part of the lung by the abundant râles.

SACCULAR BRONCHIECTASES

Saccular bronchiectases are spherical or oval dilatations which are usually confined to a definite portion of the bronchial tree. Several larger and smaller bronchiectases are generally found; the larger are usually not bigger than a cherry or, at most, a walnut. The bronchus passes suddenly or gradually into the dilatation, and it is often obliterated so that the bronchiectasis forms a completely closed cavity. The wall of a saccular bronchiectasis loses in great measure the character of the normal bronchial wall. As a rule it is atrophied to a high degree, the atrophy involving not only the mucous glands, but also the muscular fibers, the elastic elements, and even the cartilages, so that the bronchiectatic cavities seem lined with nothing but a thin membrane. The occurrence of many dilated vessels in the walls of a bronchiectasis is due perhaps to an atrophy of the vessel walls and is a symptom of clinical importance (*vide infra*). In other cases, however, we find hypertrophic processes, which involve the connective tissue of the mucous membrane, and lead to band-like projections and swellings. Finally, ulcerative processes may develop on the inner surface of a bronchiectasis and attack the surrounding lung tissue, and change the bronchiectatic cavity into a typical ulcerating cavity.

Only rarely, for example in emphysema, do we find a single saccular bronchiectasis surrounded by tolerably normal lung tissue. Its origin, then, is to be referred to causes like those which have been given above for the commoner cylindrical bronchiectases. In the great majority of cases we find

saccular bronchiectases, singly or in large numbers, surrounded by indurated and contracted lung tissue. They form one of the complications of "pulmonary contraction" [fibroid phthisis], which is almost always associated with contraction of the pleura. Since Corrigan's day we have with good reason looked upon this contraction as the chief cause for their origin. By the gradual shrinking and retraction of the lung, which, as a rule, has become adherent to the costal pleura, a traction is exerted upon the bronchial walls from without to which they gradually yield. In like manner increased inspiratory efforts as well as the stagnation and pressure of the secretion work at the same time to dilate the bronchial tube, especially where the bronchial wall is abnormally yielding as a result of disease.

Ætiology.—The question of the origin of bronchiectasis is thus directly associated with that of primary pulmonary contraction. In this connection, careful inquiry into the past history of cases shows that the disease in very many instances is referable to some previous acute inflammatory condition. This often is an acute croupous pneumonia, or sometimes a broncho-pneumonia after measles, influenza, whooping cough, etc. These pulmonary affections are followed by chronic interstitial inflammation with secondary bronchiectatic developments. Inasmuch as pleurisy is so often associated with the original pneumonia, earlier observers (Laennec, for example) concluded that the disease started in the pleura, and from there extended to the lung. This view, however, cannot be maintained. Whenever the vestiges of a previous pleurisy can be demonstrated, this has been invariably either a metapneumonic pleurisy or an empyema. In some cases the evidences of bronchiectasis follow upon a pneumonia so rapidly that we may actually speak of an "acute bronchiectatic formation" (*vide* Criegern). Here we are probably dealing with circumscribed metapneumonic abscesses, which, after discharging their contents into the bronchi, become permanent cavities. In all but a small percentage of bronchiectatic cases it is possible to demonstrate an acute pneumonic origin. In these exceptional instances we have to assume that the process from its incipiency was of a chronic inflammatory nature, somewhat similar to that resulting from the inhalation of dust, foreign bodies, and the like. It is worthy of note that bronchiectasis develops oftenest in early life. Corresponding to the most frequent location of pneumonia, bronchiectasis generally occurs in the lower lobes of the lungs, less frequently in the right middle lobe, and far more rarely in the upper lobes. As a rule, the disease is unilateral, or, at least, involves one lung to a much greater extent than the other. In the less affected lung only slight secondary changes (emphysema, bronchitis) are to be found.

"Pure" cases of bronchiectasis have nothing to do with tuberculosis. Before the discovery of the tubercle bacillus there was, of course, confusion between bronchiectatic and chronic contraction of the lungs; but we must remember that, under some circumstances, chronic tuberculosis may lead to the formation of bronchiectasis, and that, on the other hand, in bronchiectatic processes there is not infrequently a secondary development of tuberculosis. There are cases in which it is hard to decide, even at the autopsy, whether an existing chronic contraction in a portion of the lung, with induration of tissue and the formation of bronchiectasis, was originally tubercular or of some other nature.

Symptoms and Course of the Disease.—The symptoms of saccular bronchiectasis are obtained in part from physical examination and in part from observation of the sputum and of the general course of the disease. If great bronchiectatic cavities lie near the chest wall, they may give the same physical signs that we shall learn to recognize later in the description of tuberculous cavities. Bronchiectases lying within the lung, however, are often devoid of definite physical signs, so that at most we may suspect them from other symptoms, such as the peculiarities of the sputum. The more abundant the formation of bronchiectases the more does the respiration lose its vesicular character and become harsh and finally bronchial. Inasmuch as there is usually a very considerable secretion of mucus, we generally find, upon auscultation, abundant medium and even coarse moist râles. But the râles, of course, vary in number and in quality, according to the expectoration and the amount of secretion. Where there are dense pleural adhesions the respiratory murmur is very feeble, and often then only coarse, indefinite bronchial râles are audible. The percussion note over the bronchiectatic portion of the lung is usually dull or dull-tympanitic, a result of the chronic interstitial pneumonia about the bronchiectasis. The whole thorax near an extensive bronchiectasis is often much retracted and distorted. In consequence the heart and the mediastinum may be greatly displaced. X-ray examination gives us very valuable information concerning the general situation of the thoracic organs.

The expectoration is, as a rule, very abundant, and it is often raised by large mouthfuls. On standing, as a result of its thin fluid character, it exhibits a distinct division into an upper frothy, mucopurulent layer, a middle mucoserous layer, and a lower purulent layer. It usually has a peculiar stale, sweetish odor, but it may be fetid. The latter characteristic is almost always associated with stagnation of the secretion. So long as the expectoration is loose and easily evacuated, it is not fetid and the patient feels well. Then a cessation of the expectoration may ensue. The patient feels poorly, there may be a slight rise of temperature, and the expectoration becomes scanty and has a stinking fetid character. Periods of improvement may thus alternate with exacerbations. It is a favorable sign when the amount of the expectoration decreases, provided that it does not become fetid and that the patient does not feel worse. Since bronchiectasis may give rise to a permanent fetid bronchitis, and since, on the other hand, as we have said, fetid bronchitis itself often leads to the formation of bronchiectasis, we can understand the manifold interrelations and transitions which the two forms of disease present.

Sometimes large quantities of secretion collect during the night or the day, and then finally the expectoration becomes particularly profuse. Certain positions or postures (such as stooping) also induce strong paroxysms of coughing. Quincke has shown that by lowering the upper part of the thorax and placing the patient in a lateral posture, one can often artificially empty a bronchiectatic cavity in the lower lobe of the lung (Quincke's procedure). This is of some diagnostic value.

The not infrequent pulmonary hemorrhages in bronchiectasis are of great clinical importance. A few red blood cells are almost invariably found in the sputum. A mucoid sputum resembling raspberry jelly is sometimes coughed up; such sputum is intimately mixed with blood. Larger hemorrhages are due to rupture of the dilated vessels (*vide supra*) in the walls of the bron-

chiectasis. They may also be due to ulcerative processes. Slight precursors may precede a severe hemoptysis. The hemorrhage may be very abundant, and it may be repeated during a long period—several weeks—so that the patient becomes extremely anæmic. Finally, the hemorrhages cease and the patient improves quite rapidly. Such attacks of hemoptysis may be repeated very often in the course of the year. When we hear comparatively well-nourished patients with lung disease say that they have suffered for years from frequent and severe hemoptyses, we can usually infer, from such statements, that there is a saccular bronchiectasis.

The whole course of the disease is very variable, but it usually lasts for many years. It is doubtful whether a saccular bronchiectasis can actually be

FIG. 41.—Drumstick fingers in a case of bronchiectasis (personal observation).

cured; but if no more serious sequelæ occur, many patients may live for years in a tolerable condition, and may even reach an advanced age. The general nutrition often remains very good, although a certain very characteristic, anæmic, cyanotic, pale tinge of the skin gives even to corpulent patients an unmistakable morbid appearance. A very characteristic sign, present in nearly all cases, is that the terminal phalanges of the fingers gradually become clubbed and thickened, and the nails are much bent, and often have a peculiar glossiness (see Fig. 41). The cause of these changes is to be sought for in the action of absorbed toxic and septic material. On this basis, the attempt has even been made to explain the extensive, chronic bone and joint hyperplasias (*"ostéoarthropathie hypertrophiante"* of P. Marie) observed in bronchiectasis. Acute "rheumatoid" joint swellings also are not very rarely seen in this disease.

Complications.—Intercurrent febrile complications from secondary acute pneumonic processes, pleurisy, gangrene, and the like, sometimes set in.

Such complications may frequently recur in the course of years, until they finally lead to an incurable and serious state. Some patients are also in danger because of their hearts (secondary hypertrophy of the right ventricle), and œdema may set in. Amyloid disease of the internal organs, especially the kidneys, develops in some cases. Finally, metastatic brain abscess should be mentioned as a rare complication.

Diagnosis.—In most instances the diagnosis of the chronic pulmonary affection, as such, is easy. An exact conclusion is sometimes difficult, however, especially as regards a differentiation from chronic pulmonary tuberculosis. The special features to take into consideration are the general appearance, the clubbed fingers, the nature of the sputum (absence of tubercle bacilli), the seat of the disease in the lower lobes of the lungs, and the general course of the disease. A careful anamnesis is important.

We will refer to the appropriate chapters for the relations of bronchiectasis to fetid bronchitis, pulmonary gangrene, etc.

Treatment.—Treatment should provide first of all for the best possible hygienic conditions (i. e., rest, fresh air, nourishment, and climate), and also should be directed toward the amelioration of the secretion and expectoration. To diminish the secretion and to prevent or lessen fetid decomposition of the same inhalations of turpentine, Curschmann's carbolic mask and similar measures are especially recommended. The internal administration of creosote preparations (guaiacol, sirolin) is often of advantage. In addition to the usual expectorants, we may try mechanically to assist the discharge of the secretion by means of Quincke's inclined posture. All complications (hemorrhages, secondary pneumonia, pleurisy, empyema) should naturally receive special treatment. The drainage of bronchiectatic cavities by surgical means has been repeatedly attempted, but the results have not as yet proved particularly encouraging. [See remarks on page 339.]

CHAPTER VII

STENOSIS OF THE TRACHEA AND BRONCHI

(Tracheal and Bronchial Stenosis)

TRACHEAL STENOSIS

Ætiology.—Stenosis of the trachea may be caused either by diseases in the vicinity of the trachea, or by diseases of the trachea itself. The first-named mode of origin is the more frequent. To this are due all the stenoses of the trachea from compression. Enlargements of the thyroid gland from simple struma and new growths, aneurisms of the arch of the aorta and of the innominate artery, tumors and abscesses in the anterior mediastinum, swelling of the lymph-glands at the bifurcation of the trachea, abscess on the anterior surface of the cervical vertebræ, etc., may exert so great a pressure on the trachea from without that its lumen is made narrower. Besides the direct action of pressure, in most cases, a gradual atrophy and a softening of the rings of cartilage, due to pressure, sometimes play an important part, according to Rose, in the occurrence of stenosis. A collapse of the trachea may arise

from this "flaccid softening," which may come on quite suddenly, and may cause many of the cases of sudden "death due to goiter."

Changes in the trachea itself leading to stenosis are quite rare. Cicatricial stenosis as a result of syphilitic ulcerations is relatively the most frequent. New growths in the trachea are also to be mentioned, such as polypi and cancer, the latter almost always having invaded the trachea from the adjacent parts. Very rarely acute and chronic inflammatory processes such as perichondritis lead to a swelling of the mucous membrane sufficient to cause stenosis. In conclusion, we may mention that stenosis of the trachea may be due to the presence of foreign bodies.

Symptoms.—If the stenosis is so extreme that there is a real hindrance to respiration, a very striking modification of the breathing occurs. It is difficult and labored, and is performed only by the help of the accessory muscles. Both expiration and inspiration are protracted, long drawn, and accompanied by a loud stridor. In many cases inspiration is more difficult than expiration, so that there is accordingly a preponderating inspiratory dyspnœa, and the number of respirations a minute is diminished. If the entrance of air into the lungs is incomplete in spite of the lengthening of the respirations, we see an inspiratory retraction of the lower part of the thorax, and sometimes of the throat and the supraclavicular fossæ. In tracheal stenosis the larynx, however, shows little or no to-and-fro movement on respiration. This fact is of value in diagnosis in distinguishing tracheal from laryngeal stenosis, for in the latter the respiratory movements of the larynx are quite well marked.

We sometimes notice in the pulse during inspiration a marked fall in tension and in the height of the pulse waves, the *pulsus paradoxus*. With the sphygmograph we can show still more plainly the changes in blood pressure, which vary quite markedly with the respiration. The frequency of the pulse is usually a little increased, but sometimes it is diminished.

The symptoms of the disease just described may form so characteristic a picture that we can recognize it at the first glance. More precise information as to the seat of the stenosis, or the accurate differentiation of tracheal stenosis from the very similar picture presented by laryngeal stenosis, demands a direct laryngoscopic examination of the larynx and trachea, which, of course, is hardly practicable in a patient with a high degree of dyspnœa.

BRONCHIAL STENOSIS

Ætiology.—Narrowing of a primary bronchus, which is the only form to be mentioned here, arises most frequently as a result of the presence of foreign bodies—e. g., bits of bone, plum stones, buttons, etc. These may enter the air-passages by means of a deep inspiration, especially while eating or during sleep. We know that foreign bodies get into the right bronchus, which is wider, somewhat more frequently than they do into the left. Stenosis of the main bronchi from pressure also arises from aneurisms of the aorta, mediastinal tumors, enlarged bronchial lymph-glands, etc. Stenosis of the left bronchus from the pressure of the greatly dilated left auricle has been observed in mitral stenosis. Direct narrowing of the larger bronchi most frequently results from bronchial carcinoma.

Symptoms.—The symptoms are not equally distinct in all instances. They depend upon the shutting off of the corresponding part of the lung. The dyspnœa is usually very evident, especially in acute cases. The respiratory excursions are much less on the affected side than on the sound side. The percussion note remains clear; sometimes it is very deep, because the lung beyond the narrowed bronchus is constantly inflated. Auscultation shows complete absence of the vesicular respiratory murmur. We either hear nothing or we sometimes hear over the whole side a loud whistling or humming sound, the vibration of which can in some cases be felt by the hand applied to the chest wall. The vocal fremitus is diminished on the affected side. A vicarious emphysema soon develops in the other lung.

Lobular pneumonia frequently develops as a result of the entrance of foreign bodies into a bronchus, because the agents of inflammation have entered at the same time with these bodies, and, as the expectoration can be evacuated only with difficulty, and hence is more or less stagnant, the germs can readily establish themselves in it. In stenosis from pressure the condition may of course be modified in many ways by the primary disease.

PROGNOSIS AND TREATMENT

The prognosis and treatment of tracheal and bronchial stenosis depend entirely upon the nature of the primary disease. General statements as to treatment, therefore, need not be given here. A direct mechanical treatment of tracheal stenosis in appropriate cases, such as cicatricial stenosis, may be undertaken according to the different modes of dilatation above enumerated. The methods for removing foreign bodies from the larger air-passages belong to the domain of surgery. The employment of an emetic has met with distinct success in such cases, but it is not without danger, for the foreign body may wedge itself into the glottis during the act of vomiting and occasion danger of instant suffocation.

CHAPTER VIII

BRONCHIAL ASTHMA

(*Exudative Bronchiolitis. Asthmatic Bronchiolitis*)

Definition and Causes of the Disease.—Bronchial asthma is the name we give to a morbid condition whose chief symptom is a special form of severe dyspnœa. The characteristic features of the dyspnœa of bronchial asthma are, first, that it occurs in paroxysms, or at least that it grows worse paroxysmally, and, second, its peculiar manner of onset. Every case of true bronchial asthma is due to an extensive and usually quite sudden contraction of the smaller and smallest bronchial branches. This always gives rise to a form of distress for breath which differs materially from every other form of dyspnœa.

The question as to the origin of bronchial asthma coincides, then, with that of the origin of diffuse stenosis of the bronchioles. Earlier authors, especially Trousseau and Biermer, explained the suddenly developing stenosis of the lumen of the bronchioles on the basis of a tonic cramp of the circular mus-

culature of the smaller bronchi. Aside from the fact that such a spasm has never been directly demonstrated, and is also not to be easily explained theoretically, this view-point seems to me to be furthermore unsatisfactory, because it does not explain the peculiar appearance of the sputum during and after an asthmatic attack. On the other hand, the characteristics of the sputum, which will be described in detail below, point with great certainty to a special morbid process affecting the mucous membrane of the bronchioles. The frequent recurrence and the ofttimes brief duration of individual asthmatic attacks render impossible the supposition of true inflammatory disease. We must, therefore, accept the theory of a purely secretory disturbance which is either located in the mucous membrane and its secretory cells alone, or is dependent upon nervous influences. Favoring the nervous nature of many cases of asthma is, first, the fact that the disease is not infrequently observed in persons of a decided general neuropathic constitution, and secondly, that an asthmatic attack is sometimes excited by reflex action. There are, for example, some asthmatics whose attacks are induced by certain odors and inhalations (ipecac, violets, roasted coffee, etc.). Pathological alterations of the nasal mucous membrane (hypertrophy of the turbinate bones, polypi, chronic catarrh) are sometimes found in asthmatics; irritation of such lesions may even lead to an artificially induced asthmatic attack, while their removal may result in a disappearance of the attacks. Some physicians, in fact, have been inclined to refer most cases of bronchial asthma to a primary disease within the nasal cavities. In this there has been great exaggeration, and yet the possibility of asthma originating "from the nose" cannot be disputed. It is very doubtful if asthma can be caused by reflex stimuli from other organs. The asserted association of asthma with diseases of the pharynx (hypertrophy of the tonsils) is deserving of the most credence. But one should be very skeptical in accepting statements concerning the occurrence of asthmatic attacks in cases of diseases of the ear, the stomach ("dyspeptic asthma"), the intestine, the female sexual organs, etc. In these conditions, true asthma is usually confused with other dyspnœic conditions (hysterical asthma, cardiac weakness, and the like).

A reflex origin cannot be demonstrated in the majority of cases of asthma. Apparently the asthmatic attacks and the underlying acute swelling of the mucous membrane of the bronchioles begin spontaneously, or, at most, originate through certain external influences, such as the inhalation of cold air, dust, etc. If the attack is only of brief duration, and is followed by an entirely normal condition of the lungs and bronchi, we then speak of a purely nervous form of bronchial asthma. Not infrequently, however, asthmatic attacks are combined with a permanent inflammatory and catarrhal condition of the finer bronchi. The relations between asthma and bronchiolitis are not always the same. Sometimes chronic bronchiolitis only develops gradually, a long time after the asthmatic attacks have existed. Under such circumstances, we are dealing with a secondary, chronic, inflammatory disease on the basis of a specially predisposed mucous membrane. In many cases, however, we appear to be dealing with a particular form of primary, chronic disease of the bronchioles, in which the characteristic asthmatic attacks are to be considered as exacerbations of the continuous chronic morbid process. Curschmann, to whom we are indebted for the first accurate clinical studies of this form of

bronchial asthma, terms the underlying bronchial disease "exudative bronchiolitis." We, however, prefer the name "asthmatic bronchiolitis" for this condition. By these terms, a peculiar disease of the finer bronchi and the bronchioles is designated. It is characterized by the peculiar appearance of the sputum and by intercurrent, characteristic "asthmatic" attacks. Very probably, reflex nervous influences, too, favor the onset of individual asthmatic attacks in asthmatic bronchiolitis. Here, again, I am inclined to attach far greater importance to the varying amount of swelling of the bronchial mucous membrane than to a possible tonically contracted state of the musculature of the bronchioles. Some authors have also found an ætiology for asthmatic dyspnœa in spasm of the diaphragm. This assumption is, however, without facts to support it, and is directly refuted by the demonstrable fact (shown also by the X-ray examination) that the diaphragmatic excursions are unembarrassed during asthmatic attacks. Inhalations of dust (wool dust, flower dust, and the like) sometimes play a demonstrable rôle in the causation of asthmatic bronchiolitis. In addition, previous attacks of acute bronchitis must be especially taken into consideration. Some cases are referable to a severe bronchitis complicating measles or whooping cough during childhood.

The "pure" cases of asthma, which owe their origin to a special predisposition of the mucous membrane to acute swelling, acquire a general pathogenetic interest because of similar processes that occur in other mucous membranes and tissues. Thus, a certain analogy may, for example, be drawn between bronchial asthma and urticaria, as well as between bronchial asthma and certain secretory disturbances of the intestine (mucous colitis, *q. v.*). All such anomalies may be considered as the expression of a peculiar pathological tendency to exudative disturbances (the "exudative diathesis" of Czerny). Indeed, a careful investigation will not infrequently demonstrate various manifestations of this "diathesis" in different organs of the same individual. Perhaps the observations of artificial urticaria in asthma, made by Lenhartz and myself, are associated with this "diathesis." That is, we have noticed that mechanical irritation of the skin (with the handle of a percussion hammer, for example) not uncommonly gives prompt rise to an active reddening and urticaria-like wheals along the line of irritation. The possible association of this "exudative diathesis" with the general neuropathic condition is of interest, but as yet quite obscure.

Symptoms and Course of the Disease.—We will begin the account of the symptomatology by a description of the asthmatic "attack." In its purest form "nervous" bronchial asthma consists, in fact, of single attacks of dyspnœa, differing in frequency and duration, which occur at least without special cause or without any discoverable reason in persons otherwise apparently healthy. In the intervals between the attacks the patient is perfectly well, and, in particular, shows no signs of disease of the respiratory organs. In most cases of "bronchial asthma," however, the attacks, as we have said, are only more or less sudden exacerbations of a condition which in the intervals is not perfectly normal. While ordinarily only the signs of a chronic bronchitis are present, often associated with pulmonary emphysema, from time to time exacerbations occur, usually in the form of long-continued asthmatic dyspnœa, lasting for days or weeks. It is this latter form especially which can be explained only by the assumption of a true bronchiolitis.

The asthmatic attack either begins quite suddenly, or is preceded for a shorter or longer period by prodromata. These consist in a general feeling of discomfort, in abnormal sensations in the larynx or epigastrium, sometimes in remarkably frequent gaping, and often in a marked coryza associated with a good deal of secretion and frequent sneezing (compare the relation between many attacks of asthma and diseases of the nose, stated above). The attack begins in most cases at night (before midnight). The patient wakes up with an intense feeling of pressure and anxiety. Sometimes he complains of a feeling of pain in the chest. He has to sit up straight, and in severe cases even to get out of bed. He often hurries to an open window in order to "get air." His expression is anxious; his skin becomes pale and cyanotic, and sometimes is covered with a cold sweat. On objective examination we are at once struck by the characteristic change in the respiration. Both inspiration and, especially, expiration are almost always accompanied by a high-pitched whistling sound, audible at a distance. Both respiratory acts are labored, requiring the aid of the accessory muscles. On inspiration, only the upper part of the thorax is elevated to any extent. We see in the neck the inspiratory contraction of the sterno-cleido-mastoids, the scaleni, etc. Still more striking, however, is the labored, panting, long-protracted expiration, during which the abdominal muscles are contracted to a board-like hardness. We therefore recognize the disturbance of respiration in asthma as essentially an expiratory dyspnœa. The frequency of respiration is in many cases normal, or even somewhat diminished, yet we have repeatedly counted thirty or forty respirations a minute.

On physical examination of the lungs during the paroxysm, we find the percussion note normal or even strikingly loud and deep—the "box-tone." The lower boundary of the lung is usually found one or two intercostal spaces lower than normal. Not only is this condition shown in the cases with a permanent pulmonary emphysema, but during the asthmatic attack itself there occurs an acute inflation of the lung. The latter is probably explained by the fact that the lung is much stretched by the strenuous inspirations which are made by aid of the accessory muscles, while the weaker expiratory force is not enough wholly to drive out the air again through the contracted bronchioles. Therefore it happens in bronchial asthma, as in every other bronchial disease, that the expiration is usually more labored and protracted than the inspiration. On auscultation, high-pitched whistling and creaking sounds, which quite obscure the vesicular murmur, are heard over most of the lung, especially during the long expirations. In many places, indeed, where the bronchioles are almost completely closed, the respiratory murmur is entirely absent, or we hear only a low whistle on expiration. Toward the end of the paroxysm the noises become deeper and more booming, and sometimes we hear a few moist râles.

In the intervals between individual attacks, the respiratory murmur, in the cases of "pure nervous asthma," is quite normal. In cases of asthmatic bronchiolitis, even during the intervals, there are abnormal auscultatory signs (isolated bronchial râles, suppressed and weakened inspiration, prolonged expiration, etc.).

In brief paroxysms there may be scarcely any cough or expectoration. In most, particularly in the tedious cases of true asthmatic bronchiolitis, there is,

however, a scanty tough mucous expectoration. In this are found, besides the ordinary constituents of simple bronchitis sputum, larger or smaller numbers of characteristic clumps. These may be yellow or greenish-yellow, or, on the other hand, gray. The yellowish masses, which are usually very tough, and often consist of a bunch of thready matter, represent swollen and fatty-degenerated pus corpuscles, between which are very frequently interspersed a considerable number of pointed octahedral crystals. These crystals were first described by Leyden in the sputum of asthmatic patients, and are usually termed asthma crystals (see Fig. 42). Chemically they are identical with "Charcot's crystals," which are found in the leukæmic spleen, the bone marrow, etc., and they probably represent the phosphoric-acid salt of an organic base (Schreiner's base, C_2H_5N), although this has lately been questioned. As soon as the paroxysms cease the number of crystals in the sputum usually begins to diminish rapidly, and it is often possible to observe in them evident signs of disintegration. Nothing is known as to the origin of these crystals. Their presence probably bears some relation to the eosinophile cells of the sputum (*vide infra*). At any rate, Charcot's crystals are also found wherever degenerated eosinophile cells are to be seen. Not infrequently, also, numerous ciliated epithelial cells are found, in addition to the crystals, in the yellow masses. The gray plugs in the sputum of asthmatic patients consist mainly of clumps of thready mucus, and contain the peculiar "spirals" which were first described by Ungar and by Curschmann. Many of these spiral threads are visible to the naked eye, but others demand the microscope for their recognition,

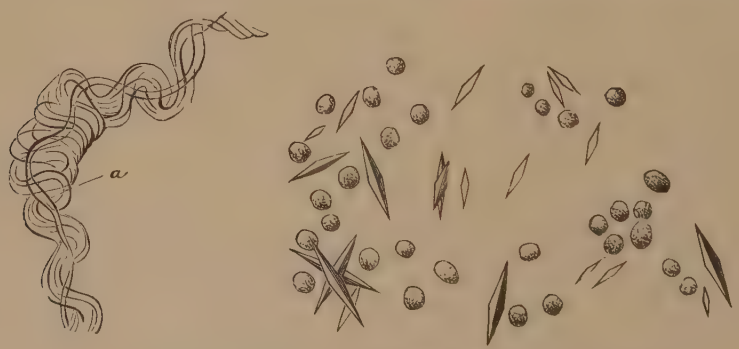

Fig. 42.—Asthma crystals and Curschmann's spirals (*a*, central fiber).

through which they are seen as brilliant forms composed entirely of various sized bands and threads collected in spirals (see Fig. 42). Sometimes a brilliant central thread of small diameter is seen in the midst of the spiral. Around the spirals are found round cells, drops of fat and myelin, and sometimes ciliated epithelium, and epithelial cells from the pulmonary alveoli. As to the precise way in which the spirals and their central thread develop, the question is not yet settled, but it is certain that the spirals represent casts of the minutest spirally twisted branches of the bronchi, and therefore clearly indicate the existence of a peculiar disease of the finest terminal bronchial twigs. The origin of the central thread is to be sought, perhaps, in the expulsive cough.

Of the other peculiarities of the sputum in bronchial asthma we may mention, first of all, the almost invariable occurrence of very many eosinophilous cells in the sputum, and also, apparently, in the blood (see Fig. 43). The significance of this fact is still unknown. We occasionally find in the sputum of asthmatics crystals of calcic oxalate and calcic phosphate.

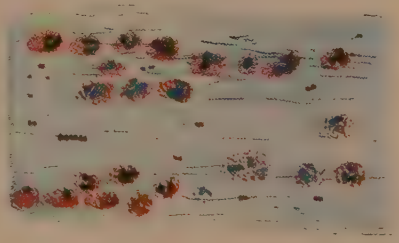

Fig. 43.—Eosinophile cells in the sputum in bronchial asthma.

The pulse is usually accelerated during the asthmatic paroxysm, the arteries contracted; the bodily temperature is normal, or sometimes even subnormal. In protracted attacks we have repeatedly seen a slight febrile movement up to about 102° F. (39° C.).

The duration of the asthmatic paroxysm is very different in individual cases, as has already been said. Sometimes it lasts only a few hours, but sometimes it lasts several days, and even weeks. Marked exacerbations and remissions of the disease usually alternate. The frequency of the paroxysms in ordinary asthma also varies exceedingly. Sometimes they come on almost every night, and then there are long pauses of months and years, so that we cannot make any general statements as to the course of the disease. Many asthmatic patients make very remarkable statements as to the individual exciting causes of their attacks. Many patients claim, for example, that the attacks occur only in certain places, while in other places they are wholly free from the trouble, that they can live only in the upper stories of the house, etc. Such statements should not remain unheeded, although it is certain that they are often due to imagination. In mild cases, cures are not infrequent; the possibility of recurrences must, however, always be remembered. Only rarely can a complete cure of the severer cases of asthmatic bronchiolitis be expected, though a decided improvement can often be obtained. In asthma of long standing, chronic pulmonary emphysema with its sequelæ almost invariably develops. The occurrence of a secondary pulmonary tuberculosis has also been repeatedly observed.

Diagnosis.—The diagnosis of the bronchial asthmatic condition as such is not difficult if we limit ourselves strictly to the characteristic type of the disease: the strenuous respiration with wheezing that can be heard at a distance, the labored, prolonged expiration, the characteristic physical signs in the lungs, and the peculiar sputum. Usually we can easily distinguish bronchial asthmatic dyspnœa from cardiac asthma (*q. v.*), spasm of the glottis (*q. v.*), and also from hysterical dyspnœa with its superficial and very rapid respiration and normal conditions in the lungs. This latter distinction of true bronchial asthma from "hysterical asthma" is very important both in prognosis and treatment, since the two are frequently confounded. If we are sure that we are dealing with true asthmatic attacks, the next question is whether it is a "purely nervous" reflex asthma or an asthmatic exudative bronchiolitis. Here, of course, we can decide only after a careful and thorough examination of the patient (especially of the nasal cavities) and after observing the course of the disease. Finally, we must also consider the possibility of a purely symptomatic asthma in chronic pulmonary emphysema, in the chronic bronchitis of renal

disease or arthritis, etc. We must, however, confine our use of the term "symptomatic bronchial asthma" to those cases in which we actually have a dyspnœa with all the characteristic peculiarities of true bronchial asthma.

Treatment.—In every case of asthma the first thing to be thought of is whether there is not a definite cause whose removal may cure the disease. In this connection we should examine the nose carefully, since numerous recent observations have shown that a previously existing asthma may wholly disappear or at least be materially relieved after the treatment of some nasal disease which may be present, such as the removal of polypi, the destruction of the erectile bodies by the galvano-cautery, etc. Of course these results should not be overestimated. It has repeatedly been claimed by nasal specialists that in many asthmatic patients we can find a definite spot in the nose and excite an asthmatic attack by touching it with a probe. This spot must then be treated first of all. We will not wholly reject these statements, but we are somewhat skeptical in regard to them. At any rate, the nose should be treated, in our opinion, only when it presents actual morbid changes.

If we cannot satisfy the causal indication in this way, we should always try next a remedy which must pass for a direct specific against certain forms of asthma—iodid of potassium. In doses of 20 to 45 gr. a day (gm. 1.5 to 3.0), or even more, if necessary, this usually causes a rapid improvement, which of course is not always, although it is frequently, permanent. In asthmatic bronchiolitis, especially, iodid of potassium in large doses often acts excellently. It seems to make the tough secretion more fluid, to ease the expectoration, and in this way to lessen the stenosis of the bronchioles. Of course a permanent and complete cure is only rarely obtained from iodid of potassium. Next to potassium iodid, I consider an energetically pursued methodical diaphoretic treatment the most effectual therapy. By the use of the electric-light baths, as well as by the other generally employed diaphoretic agents, I have usually obtained good results, and occasionally, even apparent cures in cases of nervous asthma and of asthmatic bronchiolitis. The patients are placed daily in the electric-incandescent-light bath, in which they remain from ten to fifteen minutes at the beginning of the treatment, and later from twenty-five to thirty minutes. The incandescent-light bath is followed by an ordinary warm bath, after which the patients rest for one or two hours. In many cases, after four or five treatments, a decided improvement is already observed. The attacks cease, breathing becomes freer, and the respiratory murmur clearer. An apparently complete cure may be attained in three to four weeks. If iodid of potassium and the above diaphoretic treatment have been used in vain, we must turn to the other remedies which have been employed against asthma, although their action is often quite uncertain. We may mention here the nitrite of sodium (2 parts in 120 of water, two to three teaspoonfuls a day) and nitroglycerin, which has an analogous action (20 drops of a one-per-cent alcoholic solution in ℥vjss. (gm. 200) of water, a tablespoonful two or three times a day); also pyrenol, bromid of potassium, belladonna, atropin, etc. In some cases pneumatic treatment, such as the inhalation of compressed air, has been successful, and sometimes, too, electricity (galvanization and faradization of the neck), or hydrotherapy, has been claimed to give relief. Change of climate may sometimes be of distinct service. Many patients bear the sea-air well, while with others a mountain residence exerts a favorable influence. Spe-

cial resorts (Marienbad, Kissingen, ferruginous baths, etc.) may often be recommended with advantage if appropriate to the patient's general constitution.

In severe cases a special symptomatic treatment of the attack itself is often necessary. Narcotics are without doubt the most effective, especially chloral and morphin. In severe attacks we cannot avoid injections of morphin, but we must always be cautious in order that the patient may not form the habit of using this to excess. Chloral hydrate (gr. xv to xxx [gm. 1.0 to 2.0]) often relieves the individual attack. Among other symptomatic remedies we may mention mustard plasters to the chest and calves, putting the hands and feet into hot water, inhalations of nitrite of amyl, turpentine, chloroform, pyridin, etc. Fumigation with saltpeter paper—unsized paper dipped in a concentrated solution of nitrate of potassium and dried—and the stramonium cigarettes to be had in most drug stores, are much praised. The patient may also smoke with occasional benefit stramonium or belladonna leaves which have previously been dipped in a solution of saltpeter and then dried. Many patients praise highly the different asthma cigarettes and smoke-producing powders or pastilles, which are sold as secret remedies. We, ourselves, usually employ a mixture of equal parts of powdered stramonium leaves and powdered nitrate of potassium.

[Potassium iodid is more likely to prevent recurrence if it is given continuously, for several months, at least, and it should not be thrown aside as useless until it has been pushed to the limit of toleration without avail. A convenient form of administration is in saturated aqueous solution, a minim of which represents about a grain of the drug.

The sirup of hydriodic acid may be substituted for potassic iodid in cases of intolerance of the latter. Grindelia robusta, a drachm of the fluid extract three or four times a day, serves sometimes to prevent recurrence of attacks. Marked alleviation of the paroxysms is often obtained from the inhalation of 15 to 30 drops of the iodid of ethyl.]

SECTION IV

Diseases of the Lungs

CHAPTER I

PULMONARY EMPHYSEMA

(Alveolar Ectasis. Volumen pulmonum auctum)

Nature and Ætiology of the Disease.—Pulmonary emphysema, the abnormal inflation and dilatation of the lungs, either develops in separate parts of the lung, in which case it is subordinate to other pathological changes which coexist in the lungs, or involves almost the whole extent of both lungs, and then presents the symptoms of a characteristic affection, which it is usually easy to recognize.

Attention, however, should be called to the fact that emphysematous expansion of the lungs is usually a secondary condition, developing as a sequel

to previously existing diseases. Pulmonary emphysema is by no means common as a primary affection.

The essence of pulmonary emphysema, the condition from which most symptoms are immediately derived, is the loss of elasticity in the lungs. If we compare the sound lung, with its normal elastic force, to a new and very tense rubber band, the emphysematous lung must be compared to an old and lax band that is stretched and pulled out. We therefore see why the emphysematous lung takes up a greater space than the sound one, for, on account of its lack of elasticity, it can no longer contract to its former volume. We may therefore call emphysema a permanent inspiratory distention of the lung from which it can no longer return to its expiratory condition. If we open the thorax of a subject with normal lungs, they contract, as is well known, but the emphysematous lungs remain in their inflated condition after the thorax has been opened.

If we inquire into the factors which cause this loss of elasticity in the lung, we find that they are the same kind of influences which tend to diminish the elasticity of any other elastic body. As a rubber band, by much pulling and stretching, gradually gets longer and less elastic, so the lungs, as a result of their abnormally frequent and severe distention, gradually become inelastic and emphysematous. Emphysema in many cases is a true wearing out of the lung. The normal traction of inspiration, which is continually making new demands on the elastic powers of the lungs, finally leads to a loss of elasticity in them. In advanced age most lungs become more or less inelastic. The lungs of an old man are like an elastic band which has done its work for years but which has finally given out. We therefore class emphysema of the lungs in old age rather among the states of involution, such as develop in almost all organs in advanced life, than among special pathological changes. We distinguish, moreover, most lungs with senile emphysema from other emphysematous lungs by the fact that their volume as a whole is not increased, but is rather diminished below that of the healthy lung, since we find in them the extensive atrophic processes of old age.

The condition becomes pathological, however, if the elasticity of the lung is deficient in earlier years and independently of the action of the special injurious influences which will soon be mentioned. In such cases of emphysema, developing in middle life or even in youth, the idea of a congenital weakness of the elastic elements in the lungs cannot be set aside. It probably consists in a quantitative or a qualitative defect of the elastic tissue. Some observations seem to justify the statement that a disposition to emphysema may be present in several members of the same family.

If a lung whose elasticity is previously subnormal cannot persistently satisfy the ordinary demands upon it, a normal lung, on the other hand, also loses its elasticity if the demands made upon it are greater than it can perform. This is the reason why pulmonary emphysema is in some instances to be regarded as a vocational disease. We mean here not only those influences which lead to chronic bronchitis and thus later to emphysema (*vide infra*), but more especially the abnormal demands upon the lungs in all those callings which necessitate severe physical labor. We must not only regard the deeper and more rapid respirations, but also the increased pressure during expiration to which the lungs are often exposed in the raising of heavy weights, etc. This

explains the common occurrence of emphysema in the laboring classes, and also its greater frequency in men than in women. Besides this, we must add that in certain callings, such as glass-blowing and horn-blowing, the overstraining of the lungs is much more direct. In all such cases emphysema may be termed simply a premature exhaustion of the lungs.

In very many cases—in most cases, I might say—emphysema develops as a result of chronic bronchitis. Dry catarrh of the medium-sized and finer bronchi when of long duration leads, as a rule, to pulmonary emphysema. The abnormal mechanical influences to which the lungs are thus exposed act both in inspiration and in expiration. Since the entrance of air to the alveoli is rendered more difficult by the swelling of the mucous membrane of the smaller bronchi, abnormally deep and strong inspirations are necessary, with a marked expansion of the alveoli, in order to draw a sufficient quantity of air into the alveoli. The alveolar walls are therefore exposed to an abnormal traction at each inspiration. On expiration, a pressure from within, which is perhaps even more injurious, acts on the alveoli. The ordinary expiration, which usually needs only the elastic power of the lungs, is not sufficient in chronic bronchitis to drive the air out of the alveoli through the narrowed bronchi. Thus arise the difficulty and delay in expiration which are present in chronic bronchitis, and which lead to the active participation of the muscles of expiration, the abdominal group of muscles. On forced expiration, however, the pressure does not act simply upon the contents of the alveoli, but much more upon the smaller bronchi themselves. The channel of exit for the air from the alveoli, therefore, becomes still narrower. Since the air cannot at once escape, the pressure within the alveoli is raised by the efforts at expiration, and the alveolar wall is thus again abnormally expanded. The cough, which is often present in chronic bronchitis, is a further factor, which acts in a precisely similar injurious fashion. The act of coughing begins with a forced contraction of the muscles of expiration, which follows the closure of the glottis. Until the glottis opens, therefore, the lower parts of the lung especially are put under strong pressure. The air in them, which cannot escape outward, is driven into the upper parts of the lung, and there leads to expansion of the alveoli, and finally to emphysema.

We accordingly see that a number of injurious influences coöperate in the gradual development of emphysema from chronic bronchitis, and that, sooner or later, these influences have as their result the gradual dilatation of the lungs. Here, too, we must bear in mind the individual differences in the resisting power of the lungs.

Conditions precisely similar to those in chronic bronchitis occur in other diseases, and lead in like manner to pulmonary emphysema. We very often see the development of emphysema in severe and persistent whooping cough. The worst factor here, besides the existing bronchitis, are the frequent paroxysms of coughing. Many cases of pulmonary emphysema and chronic bronchitis may be ultimately referred to such a severe bronchial disease occurring in childhood. We have already mentioned, in the description of bronchial asthma, both the acute emphysema, which occurs during the attacks, and the final development of a permanent emphysema.

In conclusion, we must here consider a theory advanced by Freund, which would make the development of an emphysema dependent upon a " primary

rigid dilatation of the thorax." It is indeed conceivable that from certain pathological changes in the costal cartilages, as Freund claims, a thorax, which had become fixed in the position of inspiration, might exert a constant abnormal traction on the lungs and so give rise to an emphysema. The occurrence of this hypothetical primary disease of the cartilages, however, has up to the present time not been established. It is rather considered by the majority of authors as a secondary change, developing as a result of emphysema or else simultaneously with it. On the other hand, it is certainly remarkable that we sometimes observe in children the "emphysematous habit" of the thorax and neck, which will be more fully described farther on, and that in fact we can often discover in such children a beginning emphysema early in life. We might perhaps imagine a congenital failure of harmony between the size of the thorax and the size of the lungs, whereby the latter are from the start in constant hypertension.

Besides the already described essential or substantial emphysema, which is a special disease attacking both lungs uniformly, we distinguish a so-called vicarious or complementary emphysema. If, by any disease, certain portions of the lungs are incapacitated in their functions, the parts which remain healthy must then assume the whole business of respiration. They become excessively expanded on inspiration, and as a result they become emphysematous. Thus we see emphysema of the upper lobes in affections of the lower lobes. Emphysema of one lung is most frequently observed clinically when the other lung is extensively diseased, especially in unilateral chronic contraction of the lung and pleura, usually seen in tuberculosis. Vicarious emphysema may also be confined to quite small portions of the lung, but then it is merely of pathological and not of clinical interest.

Pathological Anatomy.—As we have seen, the actual abnormality of the lung in emphysema is not due to an anatomical change, but to a change in its physical conditions. The loss of elasticity of the lung is shown in its greater volume, in its lack of contractility, and in its persistence in a position of inspiration.

The single alveoli are, of course, just as much expanded as the whole lung, but their walls show at first no histological changes. We have here, then, a condition which Traube has called "increased volume of the lungs," and has distinguished from the "pulmonary emphysema" proper. This distinction is without doubt justified anatomically, but clinically it cannot well be maintained. As the distention is constant, the alveolar walls cannot withstand the constant traction and pressure. This leads to progressive atrophy of their tissue from pressure—that is, it leads to a real disappearance of the elastic elements of the lung. The atrophy begins quite gradually. The partition walls of the alveoli are first perforated, and then they partly or wholly break down. The neighboring alveoli run more and more into one another, and thus finally arise alveolar ectasis and infundibular ectasis, which can be made out with the naked eye, and which may attain a diameter of $\frac{1}{10}$ to $\frac{4}{5}$ of an inch (0.5 to 2 cm.) or more. If single air bubbles enter the interlobular, interstitial, or subpleural connective tissue, which may happen perhaps in severe fits of coughing, we speak of an interstitial or interlobular emphysema, in distinction from the ordinary vesicular or alveolar emphysema.

The tissue atrophy in the septa of the alveoli affects not only the elastic

tissue, however, but also the branches of the pulmonary capillaries in the alveolar walls. The affection of the elastic tissue adds no new conditions to the disturbed functions of the emphysematous lung, which we have just described. The destruction and final atrophy of the pulmonary capillaries, however, is the second important factor in the pathology of pulmonary emphysema, for, with the destruction of so great a part of the vascular area in the lungs, the outflow from the right side of the heart is considerably impeded. There must therefore necessarily be a stasis in the pulmonary arteries and the right side of the heart, and the right side of the heart can overcome the increased resistance only by increased work, and thus in chronic pulmonary emphysema there must finally arise a dilatation and consecutive hypertrophy of the right ventricle with their further consequences.

Symptoms and General Course of the Disease.—Although a pulmonary emphysema may sometimes, as in whooping cough, develop in a comparatively short time, still, its course is always very chronic. In most cases the origin of the disease is quite gradual, as in all those instances in which emphysema develops from chronic bronchitis or asthma, or as a result of some injurious occupation. The symptoms gradually and insidiously associate themselves with those of the chronic bronchitis.

The symptoms of emphysema usually begin in middle or advanced life, but marked emphysema may occur even in youth and childhood. The disease always lasts for years, unless some fatal intercurrent disease arises.

The objective and subjective symptoms are due either to the chronic bronchitis, which very often coexists, or to the emphysema itself. As we have seen above, chronic bronchitis is nearly always the primary disease from which emphysema gradually develops. On the other hand, to be sure, emphysema can lead to a stasis of the pulmonary circulation (*vide infra*), and in this manner favor the development or the increase of a chronic bronchitis. At any rate, the two diseases, emphysema and chronic bronchitis, are closely connected clinically.

Bronchitis causes its well-known symptoms—cough, expectoration, moderate dyspnœa, and a feeling of pressure in the chest. The bronchiectases, which are often gradually formed, especially in the lower lobes, may lend a peculiar stamp to the cough and expectoration (*vide* page 198). Emphysema increases the patient's dyspnœa to a degree which can only rarely be caused by chronic bronchitis alone. The emphysematous lungs soon become incapable of satisfying any extraordinary demands of respiration. Many patients are only slightly conscious of the difficulty in breathing so long as they keep quiet, but whenever they make a trifling physical exertion, go upstairs, or take a little longer walk than usual, the dyspnœa comes on.

The variations in the intensity and extent of the bronchitis correspond to the frequent and quite marked variations in the patient's feelings. These variations depend upon the condition of the patient, his circumstances, and the possibility of his taking care of himself; the change of seasons, too, has an influence on him. In pleasant weather many patients live in tolerable comfort, but autumn and winter bring an aggravation of all their symptoms with the increase in their bronchitis.

The last stage of the disease is characterized by the appearance of disturbance of compensation in the heart. We have seen above that the cause of the

impairment of the pulmonary circulation, and of the resulting hypertrophy of the right ventricle, is the closure of numerous pulmonary capillaries. A further reason for the impairment of the circulation comes from the disturbance of respiration itself, since the influence of the respiratory movements on the circulation is well known. The appearance of a marked disturbance of the circulation may be deferred for some time by the increased efforts of the right ventricle. The cyanosis of most patients, however, is due not only to incomplete oxidation but to the blood stasis which extends backward from the right side of the heart into the veins of the body. Finally, the right ventricle becomes more and more feeble, the stasis in the veins increases, œdema of the extremities and transudation into the various cavities of the body ensue, and after long suffering the patient succumbs to dropsy.

Emphysema is frequently combined in its later stages with other chronic diseases. Pulmonary emphysema with its sequelæ is seldom found at the autopsy as a single lesion, but we discover in the cadaver coexisting disease of the heart, the blood vessels, or the kidneys, all a genuine wearing out of the aging body. Pulmonary tuberculosis is often a final development in emphysema, but it is usually of the chronic indurated form, and is not very extensive.

Physical Examination. 1. *Inspection.*—In many patients we can detect the disease with considerable certainty at the first glance; we are therefore justified in speaking of an emphysematous habit. (See Fig. 44.) The patients are usually quite well nourished, at least in the early stages of the disease, and are often rather corpulent. They appear plump or even somewhat bloated, and their faces are more or less markedly cyanotic. The configuration of the neck and thorax is especially characteristic. The neck is usually short and thick; the sterno-cleido-mastoid muscles, which have to act as auxiliaries in inspiration, are tense and hypertrophied, especially during the inspiratory contraction. The inspiratory contraction of the scaleni may also be seen and felt. The veins in the neck are visibly dilated, and in severe cases are swollen to thick blue cords, and we sometimes see in them evident undulating or pulsating movements. The thorax is rather short, but broad and strikingly deep —the "barrel-shaped thorax." The intercostal spaces are narrow, and the lower ribs incline only a little downward. The epigastric angle is therefore obtuse, and sometimes becomes almost a straight line. The respiratory movements are almost always accelerated in severe cases. Inspiration becomes short and labored. The excursions of single ribs are therefore slight, and the thorax is raised rigidly and more as a whole. Expiration is distinctly prolonged. There may be a noticeable retraction of the intercostal spaces on inspiration, in the lower and lateral portions of the thorax.

This characteristic form of the thorax in emphysema is regarded as a constant inspiratory position of the ribs, and corresponds to the permanent inspiratory dilatation of the lungs. The peculiar rigidity of the thorax is probably due to the changes in the costal cartilages already described, which, according to Freund, are primary. In many cases the emphysematous form of the thorax gradually develops in the course of the disease, but in other cases it seems to depend on some original predisposition to the disease (*vide supra*).

In conclusion, we must state that the above description corresponds to the typical form of emphysema, from which we may have many deviations. In

the paralyzed thorax, for instance, we may meet with a high degree of essential emphysema of the lungs, which may give rise to errors in diagnosis.

2. *Percussion.*—Percussion gives very decided results in the diagnosis of pulmonary emphysema. We find the inferior border of the lungs one or two intercostal spaces lower than under normal conditions, corresponding to their

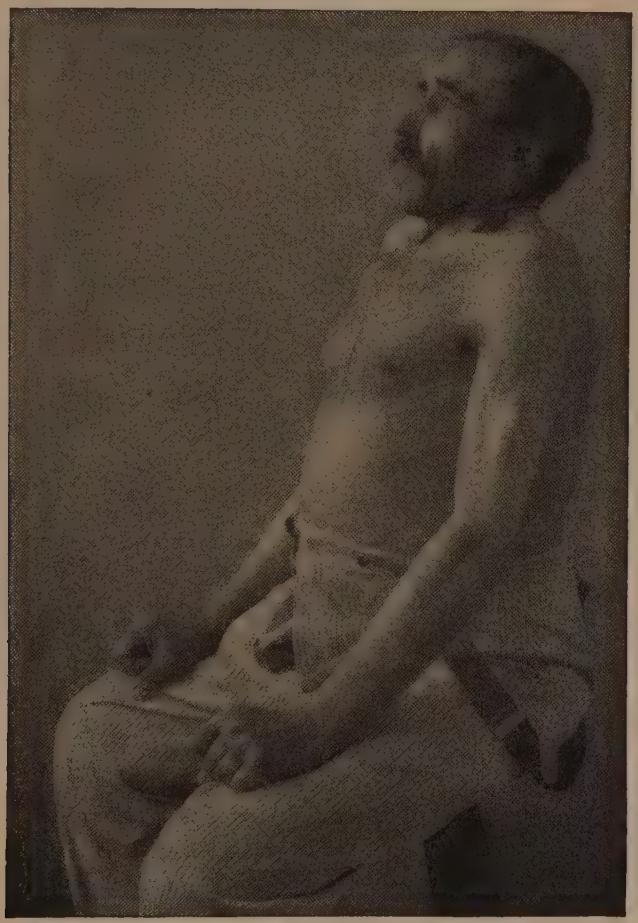

FIG. 44.—Advanced pulmonary emphysema. (Breslau Medical Clinic.)

permanent inspiratory inflation. Clear pulmonary resonance on the right front in the line of the nipple extends to the lower border of the seventh, and sometimes of the eighth, rib. On the left front it extends to the fifth or sixth rib, so that the cardiac dullness is lessened. The area of cardiac dullness can often not be made out at all; or, at most, on strong percussion, it is made out in a limited extent as relative dullness. The pulmonary resonance extends on both sides in the back to the first or second lumbar vertebra. These results of percussion in emphysema, however, are frequently altered, because other conditions, such as passive congestion of the liver, meteorism, and ascites, may be present at the same time, and push up the diaphragm. Thus the

detection of emphysema by percussion is made decidedly difficult. An increase in the lung area is also absent in the emphysema of old age (senile atrophy of the lungs).

Qualitative changes in the percussion note may be entirely wanting in emphysema. The pitch is sometimes remarkably loud and deep—the "box tone" [tympanitic resonance]; but in other cases, especially in the back, we find it somewhat dull. This may depend in part upon the poor vibratory conditions in the rigid chest walls, but in other cases it is caused by the retention of an abundant secretion in the lower lobes.

On inspiration, the lower edge of the lung moves downward very little or not at all. This is an important sign in diagnosis. Since the lung is always in a state of abnormal inspiratory distention, and since the entrance of air is impeded by the accompanying catarrh of the bronchi, the difference between the inspiratory and expiratory expansion of the lung is much diminished. The difference caused by respiration in the lower limit of the lung affords a good measure of the respiratory disturbance in the lower lobes.

The detection of dilatation and hypertrophy of the right ventricle by percussion is in many cases difficult, because the lungs cover the heart. A positive result can be obtained only by carefully defining the relative cardiac dullness. [For this purpose some prefer vigorous percussion and others very light strokes.] The epigastric pulsations frequently seen in emphysema, and also the marked undulating and pulsating movements in the jugular veins, are to be regarded as quite certain signs of dilatation of the right side of the heart.

3. *Auscultation.*—The characteristic auscultatory sign of emphysema is the prolonged expiration. As a flabby rubber band, when it is stretched and then let loose, no longer snaps back quickly and strongly, so the emphysematous lung, when it has been stretched in inspiration, comes back again only slowly. We hear with it a somewhat aspirated, sonorous sound, which plainly exceeds the vesicular inspiratory sound in duration. The vesicular murmur itself often undergoes a modification in pulmonary emphysema. It has a sharp sound, as if the breath were sucked in, or in other cases it is rougher and more indefinite. In a high degree of emphysema the vesicular respiration is sometimes very faint and obscure, because the inspiratory current of air is reduced to a small amount in the lungs, which are already excessively dilated. In many cases we hear rhonchi beside the respiratory murmur, dry whistling, buzzing, and creaking sounds on inspiration and expiration. If cylindrical bronchiectases have already formed, we hear, especially over the lower lobes, numerous fine and medium moist râles, but no sonorous rhonchi. The adventitious sounds may wholly conceal the respiratory murmur. With a marked retention of secretion we sometimes hear nothing but a low, suppressed rattling.

The heart sounds are usually rather feeble, because the heart is covered by the lung. The "functional systolic murmur of emphysema" at the apex, described by some writers, we have heard much less frequently than we should expect after the statements relating to it. If present, it is probably due to coexisting valvular changes. The pulmonic second sound in emphysema is, as a rule, markedly accentuated, as a result of the stasis in the pulmonary circulation.

The diminution of the expiratory pressure in emphysema may be measured with the manometer, or with Waldenburg's "pneumatometer." The normal expiratory pressure of 110 to 130 mm. sinks in emphysema to 100 or 80 mm. As we should expect, the spirometer shows a diminution of the vital lung capacity, which can be readily explained. The normal lung capacity of about 3,500 c.c. falls to 2,000 or 1,000 c.c.

Other Symptoms in the Lungs and in Other Organs.—In regard to the other symptoms in the lungs we have only a little to add to what has already been said. The intensity of the cough naturally varies in individual cases according to the degree of the existing bronchial catarrh. Many patients are troubled by a dry cough, while others have abundant expectoration. There is nothing characteristic of emphysema in the composition of the latter. All the kinds of sputa which are found in the different forms of chronic bronchitis are also found in pulmonary emphysema. The dyspnœa, whose predominantly expiratory character we have already mentioned, increases in advanced cases to a most marked degree. Sometimes the increase shows itself by the appearance of distinctly paroxysmal dyspnœa. This is often really to be regarded as a symptomatic bronchial asthma, but, on the other hand, we must not overlook the fact that a temporary increase of the bronchitis, retention of secretion, and cardiac failure, may also excite attacks of dyspnœa, which cannot properly be termed asthma.

The important changes in the heart resulting from emphysema have already been described. The exhausted right ventricle can no longer overcome the increased resistance in the pulmonary circulation. The difficulty of respiration is still greater, from the passive congestion of the pulmonary vessels. The skin becomes still more cyanotic, and finally œdema and general dropsy develop. The failure of compensation is indicated by the smallness of the pulse, its increased frequency, and sometimes by its irregularity; [and also by the fact that the pulmonary second sound ceases to be accentuated]. The difficulty of an objective examination of the heart in emphysema has been spoken of above.

The appearances of blood stasis in the internal organs are shown especially in the liver and kidneys. The liver is swollen, and its increase in size (the liver of passive congestion) can frequently be made out by percussion or palpation. The pains in the hepatic region, of which many patients complain, are perhaps due sometimes to the stretching of the capsule of the liver, but they are probably more often muscular pains excited by the frequent coughing.

In the kidneys the effect of stasis is first shown by a diminished excretion of urine. The urine is more scanty in amount, more concentrated, of a higher specific gravity, and of a darker color. It generally gives an abundant sediment of urates, and it may contain a small amount of albumen. Microscopically we may find a few hyaline casts, and a few red and white blood corpuscles. It is evident that this diminished activity of the kidneys favors the development of dropsy.

The spleen is not infrequently found congested at the autopsy. The evidence of this, however, is often uncertain during life, for percussion of the spleen is difficult on account of the emphysema, and palpation is difficult from the anasarca.

Gastro-intestinal symptoms may be present in emphysema. The appetite seldom remains good throughout the disease. Many patients suffer from chronic constipation; and more rarely there is a tendency to diarrhea.

Fever is not present in simple pulmonary emphysema. Whenever fever exists for a long time it depends on other complications, such as severe bronchitis, pneumonia, or tuberculosis.

Complications.—Complications of emphysema with other chronic diseases are frequent. The old opinion that emphysema and tuberculosis, and emphysema and chronic heart disease, were antagonistic to each other is entirely false. These complications are not very rare. We may also mention the complication with general arteriosclerosis and with chronic nephritis, especially the contracted kidney. Among acute diseases we must mention particularly the acute accidental pulmonary complications, to which emphysematous patients, particularly of the lower classes, are much exposed as a result of overexertion, exposure to cold, etc. Acute febrile bronchitis and lobular pneumonia cause marked exacerbations, and attacks of true influenza and croupous pneumonia in aged patients are not infrequently dangerous to life.

Diagnosis.—The diagnosis of emphysema can be made directly from the results of the physical examination, and usually presents no difficulties. We think it important merely to say that too much stress should not be laid upon a low position of the lower edge of the lung alone. Many men apparently have dilated lungs but no symptoms at all. The chief thing, therefore (besides the dilatation of the lungs), is the presence of delayed and difficult expiration and lessened power of inspiration. The last-named symptoms alone render a diagnosis of senile atrophy of the lungs (the emphysema of old age) possible. For, as indicated above, the lungs are not distended, but rather are shrunken and atrophic. When a pronounced emphysema is associated with decided thoracic symptoms, the question always arises as to whether the emphysema is the sole cause of these manifestations, or whether there is not, in addition, some disease of the heart, the blood vessels, or the kidneys (contracted kidney). The contracted kidney is generally recognizable by a careful urinary examination. It is more difficult to judge of the heart and the aorta, especially in the presence of pulmonary distention. In previous years the diagnosis of "pulmonary emphysema" was far more frequently made than at the present time, when much more attention is paid to muscular diseases of the heart and to sclerosis of the aorta, and when the X-ray examination furnishes us with a new and exceedingly valuable means for the recognition of these conditions. At any rate, Röntgen-ray examination should never be omitted in any severe case of emphysema. The diagnosis may also offer great difficulties when a patient with emphysema is not examined until the final, dropsical stage. In such cases it is often very difficult to avoid confusion with heart disease (myopathic hypertrophy, myocarditis, mitral stenosis), contracted kidney, etc. It is often difficult, also, to come to a decision in those cases in which there are evident signs of coexisting cardiac or renal disease besides the emphysema. In such cases it is often hardly possible to decide upon which of the different organic changes the chief stress is to be laid. In all such cases we must place especial confidence in an accurate history as well as a careful physical examination, including the X-rays. The special nature and the sequence in development of the individual symptoms

often afford valuable data for a correct appreciation of the whole clinical picture.

Prognosis.—Pulmonary emphysema of acute origin—that is, acute inflation of the lung—like that resulting from whooping cough and analogous affections, may be recovered from in many cases. Otherwise, as regards the final curability of the disease, the prognosis is wholly bad. The duration of the disease and the intensity of the symptoms are, of course, very different in individual cases. Here almost everything depends upon the worldly circumstances and hygienic surroundings of the patient. With sufficient care the disease may be tolerably well borne for many years, but without it the first symptoms of respiratory and cardiac insufficiency appear much earlier. The final termination is usually due to complications (*vide supra*).

Treatment.—Since emphysema itself is only slightly amenable to treatment, most of our therapeutic remedies are directed to that accompanying condition upon which the greater part of the symptoms depend—the chronic bronchitis. If we succeed in improving this, or even in wholly removing it, we always obtain a decided improvement in all the patient's symptoms. The therapeutic agents mentioned in the description of chronic bronchitis are therefore of frequent use in emphysema.

In the first place, we must seek the best hygienic conditions for the patient, and remove him from all injurious influences, such as dust, bad air, and great physical exertion. In dry catarrh we should use the alkaline mineral waters, and when there is abundant mucous secretion the balsams, such as turpentine internally and by inhalation. The most valuable expectorants are iodid of potassium, apomorphin, and ipecac when the expectoration is tough, and liquor ammonii anisatus and senega when it is abundant. A widely employed mixture ("emphysema drops") is the following: Liq. ammonii anis., tinct. stramonii, tinct. opii simpl., āā ℥ijss. (10 gm.), the dose being 15 to 20 drops three times daily. (For further formulæ, consult the Appendix.) The action of these drugs, of course, too often fails of the desired result, so that we frequently have to change our remedies. When there is a troublesome cough, disturbing the sleep, we cannot dispense with narcotics, such as morphin, codein, or Dover's powder. If severe dyspnœa comes on, we may try to obtain relief by mustard plasters to the chest, or by immersing the hands and feet in hot water. With asthmatic attacks we may try iodid of potassium, besides the other remedies mentioned for asthma. A mixture of 3 gr. (0.2 gm.) of the sodium salicylate of caffein, and 12 gr. (0.8 gm.) of antipyrin, or the stramonium preparations, may also be of occasional value. Here, too, we must finally resort to narcotics.

We must carefully watch the condition of the heart, and use digitalis when there are signs of beginning disturbance of compensation and the pulse grows small and irregular, and this drug may prove very useful. If symptoms of dropsy set in, we may sometimes prescribe diuretic remedies, such as juniper tea, acetate of potassium, diuretin, theocin, calomel, etc., besides digitalis. When there is persistent weakness of the heart, we should also use digitalis and other stimulants (strophanthus, camphor, wine, etc.).

Besides the purely symptomatic treatment thus described, the attempt has been made to meet the causal indications in emphysema, and especially to aid the patient in expiration, and thus to improve the power of the lung to con-

tract, where it is possible. To this end Gerhardt has recommended assisting expiration mechanically by compression of the thorax. This compression must be done methodically by another person,[1] about five or ten minutes every day, by the aid of both hands laid flat on the lower lateral portions of the thorax. The effect of this manipulation in diminishing the dyspnœa and making expectoration easier is in many cases very satisfactory. The "breathing chair" made by Rossbach has a similar mechanical action.

The employment of the pneumatic treatment has also become quite general, especially since the introduction of Waldenburg's portable apparatus. The expiration into rarefied air, which meets the causal indication, may procure great relief for the patient in many cases, and sometimes, too, may result in an improvement of the emphysema which can be demonstrated on physical examination. Inhalations of compressed air are also employed when there is severe bronchial catarrh. Still, too much must not be anticipated from pneumatic treatment.

CHAPTER II

PULMONARY ATELECTASIS

(Compression of the Lungs. Aplasia of the Lungs in Cases of Kyphoscoliosis)

Ætiology.—Atelectasis of the lungs is a condition the direct opposite of emphysema. While in the latter the lungs are abnormally inflated, in the former they are abnormally collapsed. The air has disappeared from the alveoli and lesser bronchi, and in the most advanced cases even from the larger bronchi. The atelectatic portions of the lung are not altered histologically, but are changed to a firm tissue, deprived of air—so-called splenization or carnification.

The atelectasis of the newborn is due simply to deficient respiration and to the consequent imperfect entrance of air into the lung. In weak children, who die soon after birth, we often find the lower lobes wholly or in part in a fetal, uninflated condition—that is, atelectatic. By artificial inflation we can readily expand the lungs to their normal extent. In many cases of weak, newborn infants there is at first an atelectasis of parts of the lung which gradually disappears entirely and is replaced by normal conditions.

Acquired atelectasis occurs in two ways. We may mention, as the first and most frequent ætiological factor, the plugging of the smaller bronchi. If a complete closure of a bronchus arises from the accumulation of secretion, as may easily happen in the narrow bronchi of children, the air can no longer enter, on inspiration, into that portion of lung supplied by the plugged bronchus. The air which is shut up in it is gradually absorbed by

[1] One of the author's patients at the polyclinique in Leipsic made a very simple but very effective apparatus for producing this compression of the thorax on himself by the aid of two narrow boards, which are fastened together at one end by a cord of suitable length. These boards, which are furnished with a pad at this end fitted to the wall of the chest, are laid flat on the two sides of the thorax so that their free ends project forward some six inches or a foot, and serve as a one-armed lever. By pressing them together the patient himself can thus, without any strain, exert a considerable pressure on his thorax with each expiration.

the blood. The adjacent parts of the lung expand, and the portion that is excluded from respiration collapses, leaving a circumscribed pulmonary atelectasis, usually rich in blood but devoid of air. Such atelectases, in greater or less number and extent, are very often found in the bodies of children who have suffered from severe bronchitis, especially after measles, whooping cough, or diphtheria. Besides the direct action of the plugging of the bronchus, the weakness of the respiratory movements and the inability to cough vigorously, conditional upon the constitutional condition, play a significant part.

The second very frequent and important cause of pulmonary atelectasis is compression of the lung. In all the diseases which diminish the space for the expansion of the lungs, the lungs are compressed to a greater or less extent, whereby the air is squeezed out of them. Thus arise the atelectases from pressure in pleuritic effusion, hydrothorax, pneumothorax, in marked cardiac hypertrophy, pericardial effusion, and aneurism of the aorta. Atelectasis of the lower lobes also arises in the same way from great upward pressure on the diaphragm by ascites, meteorism, abdominal tumors, etc.

That form of pulmonary atelectasis which arises from deformities of the thorax is of great practical importance. In severe kyphoscoliosis, the half of the thorax corresponding to the convexity of the vertebral column is much narrowed. The lungs are materially hindered in their expansion, and even in their growth, if the deformity occurs in youth. This is called "aplasia of the lungs," a condition which may give rise to grave results (*vide infra*).

Symptoms.—In the majority of cases the symptoms of atelectasis are subordinate to the disturbance caused by the primary disease. This is especially true in most of the atelectases from pressure, although the most dangerous factor lies in the compression of the lung.

The atelectasis of the lungs developing as a result of diffuse capillary bronchitis, especially in children, can, of course, not be detected by physical examination until it is of great extent. The respiration, in extensive formation of atelectasis, often shows a very striking and characteristic deviation from the ordinary type, especially when the atelectasis develops in the lower lobes. It is accelerated and labored, and is performed chiefly by the upper and anterior portions of the thorax. In the lower portions we see marked inspiratory retractions, which are caused in part by the external pressure of the air, and in part by the exaggerated contraction of the diaphragm.

Physical examination can, of course, reveal abnormal conditions, especially dullness on percussion, only when the atelectasis is extensive. Dullness, however, is usually hard to make out in children. Auscultation gives signs of existing bronchitis; and sometimes, too, with more extensive consolidation, there is bronchial respiration. In other cases, as may be easily seen, the respiratory murmur is much diminished or wholly absent. Thus, the physical signs of atelectases are not easily distinguishable from those of pneumonia, especially of lobular pneumonia. In fact, a sharp distinction between atelectatic nodules and nodules of lobular pneumonia in the lung cannot be drawn clinically.

Aplasia of the lungs in kyphoscoliosis demands a special description, because it is of great practical significance. Many patients with kyphoscoliosis

may live for years without special respiratory disturbance. Careful observation, of course, usually shows a somewhat labored and hurried respiration, but the patients have not paid much attention to it. In other cases the difficulty in breathing is more noticeable. The person affected is incapable of any severe physical exertion; he always feels short of breath, and often suffers from cough and expectoration. In the cases first mentioned, however, which for years have had little or no trouble, disturbances in respiration sometimes come on quite suddenly. They may also frequently arise without any special cause, and may attain a very threatening degree. The condition may improve, or it may lead to comparatively or even absolutely sudden death. Examination of the lungs during life usually shows nothing but the signs of an extensive bronchitis. By careful percussion we may quite frequently detect an increase of cardiac dullness to the right. Sometimes a moderate œdema develops. In such cases the autopsy shows nothing as the cause of death but the changes in the chest. The lungs are deficient in air, small, and compressed, but in circumscribed portions, on the contrary, emphysematous and expanded. The right side of the heart in the great majority of cases is dilated and hypertrophied. There can scarcely be a doubt, therefore, that the cause of the onset of severe symptoms and the final cause of death is to be sought in the cardiac failure.

Finally, it is worthy of mention that there is a frequent form of mild atelectasis in the lower lobes, which occurs in very sick and bedridden patients who usually keep in one position—on the back—as in typhoid fever. On making such patients sit up we hear during the first inspirations exquisite crepitant râles over the lower lobes, which sometimes disappear after a few deep inspirations. Here we have to do with a mild atelectatic condition, with a temporary collapse of the alveoli and smallest bronchi.

Treatment.—The treatment of atelectasis coincides in great measure with the treatment of the primary disease, and is therefore to be looked for in the corresponding chapters. The prophylaxis of atelectasis, by constant attention to the respiration, is of great practical importance. We should try to keep the patient from lying continually on his back, and we should make him take deep inspirations. The timely use of tepid baths, with effusions, is a special preventive of the development of atelectasis, and it may bring about a recovery when atelectasis is already present.

Tepid baths may also be used with care in the treatment of dyspnœa caused by kyphoscoliosis. The condition of the heart, however, deserves especial attention (stimulants and digitalis). The reader is referred to the consideration of the general treatment of circulatory disturbances in the chapters on diseases of the heart. In other respects the symptomatic treatment by expectorants, etc., is the same as in other chronic pulmonary affections.

For a long time I have entertained the impression that it might be possible to give the compressed lungs, in cases of Pott's disease, freer play by performing an extensive resection of the ribs. To my knowledge such a procedure has never been practiced.

CHAPTER III

PULMONARY ŒDEMA

Ætiology and General Pathology.—We have in pulmonary œdema the exudation of a highly albuminous fluid, usually somewhat hemorrhagic, not only into the interstitial tissue, but also into the alveoli themselves. The danger of the condition is easily understood from the high degree of dyspnœa which immediately ensues from it. In fact, pulmonary œdema is in many cases a terminal symptom, which comes on in all forms of acute and chronic disease. Many patients are said to die with the signs of pulmonary œdema, especially patients with heart disease, pulmonary and renal disease, and also with other affections of the most varied kinds.

In rare cases pulmonary œdema is a transitory symptom. Repeated attacks of it may occur, especially in heart disease and chronic renal disease, and, for a time, at least, the patient recover from them.

In spite of much clinical and experimental work, the special causes of pulmonary œdema are still quite obscure. For one class of cases the work of Cohnheim and Welch shows that pulmonary œdema is to be considered as purely the result of stasis. Pulmonary œdema takes place when the outflow of venous blood in the lung meets an obstacle which can no longer be overcome by the mechanical force of the right ventricle. The obstacle which plays the most significant part here, and which may occur in all possible forms of disease—of course more readily in those mentioned above than in others—is paralysis of the left ventricle. If the further progress of the blood is much hindered by this, the overfilling of the pulmonary circulation and a consequent pulmonary œdema will necessarily follow, in spite of the most vigorous action of the right ventricle. Many cases of terminal pulmonary œdema seem to depend upon the fact that the left ventricle is paralyzed in its action sooner than the right.

The paralysis of the left ventricle, however, is certainly not the only factor to be considered in the origin of pulmonary œdema. We must also consider the condition of the walls of the blood vessels in the lungs. In many cases, especially in renal disease, pulmonary œdema seems to depend upon local changes in the vessel walls (Sahli). This form of pulmonary œdema is somewhat akin to true inflammatory œdema. The latter is found chiefly in the vicinity of portions of lung infiltrated with pneumonia; it is usually of limited extent, and therefore it is of subordinate importance as a cause of disturbances in respiration compared with the diffuse œdema of stasis.

In very rare cases, as we have ourselves seen, an apparently primary acute pulmonary œdema, with a speedily fatal termination, develops in men who are apparently perfectly healthy, and the autopsy gives no explanation of its origin. We perhaps have to do in these cases with the sudden failure of the left ventricle, but probably with acute vascular changes as well.

Symptoms.—Marked dyspnœa is the most striking symptom in pulmonary œdema. It is subordinate only when the patient is found in the death agony and is no longer fully conscious.

In pulmonary œdema the respiration is hurried, labored, and rattling. All

the accessory muscles of respiration are called into play. The patient usually sits upright in bed. We see on his lips and cheeks a gradually and constantly increasing cyanosis, and we often hear at a distance the moist râles originating in the larger bronchi.

On examination of the lungs, the percussion is essentially normal, if there is no other disease of the lungs. Sometimes the percussion note is a little higher in pitch, and often it is slightly tympanitic. On auscultation, we hear everywhere many fine and medium moist râles. If the patient can still expectorate, he raises a large amount of frothy, serosanguineous sputum. The whole picture of the disease is so characteristic that the condition is but rarely mistaken.

Treatment.—Since in most cases pulmonary œdema is not so much the cause as a symptom of approaching death, our remedies against it are apt to prove powerless, but it must always be our duty, at least in all cases that are not absolutely hopeless, to try to relieve the pulmonary circulation. From the pathogenesis of pulmonary œdema it follows that we must pay particular attention to the condition of the heart, especially of the left ventricle. Hence we should use energetic stimulants, especially subcutaneous injections of camphor or ether, every half hour or hour. Internally we give first strophanthus (10 drops of the tincture every hour) and also camphor, wine, and very strong *café noir*. Besides that, we apply strong irritants to the chest, such as large mustard plasters or hot sponges. Sometimes a decided improvement of the respiration, when it has nearly stopped, may be obtained by a bath with cold douching. When there is marked general cyanosis, if the patient is on the whole strong and well nourished, venesection is sometimes of manifest benefit. Emetics, however, accomplish little, and are even dangerous on account of the collapse which is apt to come on after them. Expectorants (benzoin, liquor ammonii anisatus) are more frequently prescribed, and an energetic "derivation to the intestines," by senna, calomel, or enemata of vinegar, seems sometimes to be really of service. Acetate of lead in large doses, 1 or 2 gr. (gm. 0.05 to 0.10), in powder, every hour, employed empirically by Traube, is deserving of trial.

In this way, especially in acute diseases like typhoid and pneumonia, we in fact sometimes succeed in averting the danger of pulmonary œdema by rapid and energetic action. In the cases of œdema occurring in incurable chronic diseases of the heart and kidneys, the remedies employed are, of course, unfortunately incapable of preventing death.

CHAPTER IV

CATARRHAL PNEUMONIA

(Broncho-pneumonia. Lobular Pneumonia)

Ætiology.—Catarrhal pneumonia is neither ætiologically nor anatomically one single, absolutely independent disease, but from a clinical standpoint we are perfectly justified in grouping together the catarrhal, lobular pneumonias, which are usually secondary to other diseases, and especially to a previous

bronchitis, and in contrasting them with croupous, lobar, "genuine" pneumonia. In the great majority of catarrhal pneumonias the belief is certainly correct that the agents of inflammation do not enter the pulmonary alveoli directly from without, but that the inflammatory process is situated originally in the bronchi, and from this point extends downward to the special respiratory parenchyma of the lung. This extension of the inflammation may take place entirely by continuity, or it may skip some part, as the germs are often inhaled from the bronchi directly into the infundibula and alveoli. The last-mentioned parts must, however, possess considerable power of resisting germs, since the danger of an extension of bronchitis to the alveoli generally exists only in the severer and more extensive cases of bronchitis or under other peculiar conditions. This extension of the process is not uniform in all parts, but it occurs first in the distribution of some small branches of the bronchi, and this is the reason why the pneumonic infiltration affects first only a few bronchial areas—that is, individual lobules.

In opposition to this generally accepted theory of the method of origin of the true "lobular" or "broncho-pneumonic" foci, the attempt has recently been made to show that the inflammatory process can pass through the walls of a small bronchus directly into the neighboring parenchyma of the lung, and that it may then extend farther along the lymph-channels. Clinically, however, we cannot yet distinguish this form of focal pneumonia from the ordinary catarrhal pneumonia.

If we ask what are the conditions under which we are most apt to observe the development of lobular pneumonia, we must first mention a number of acute infectious diseases, in which the air-passages are primarily affected, or in which they may very readily be involved sympathetically. Chief among these are measles and whooping cough, and next diphtheria, influenza, smallpox, etc. In all these diseases there is either a bronchitis at the beginning, or else it can develop very easily in them. It is in these same diseases, also, that simple bronchitis comparatively often develops into lobular pneumonia.

Furthermore, in almost all severe acute and in many chronic diseases the conditions favor the development of secondary bronchitis and subsequently, at times, of lobular pneumonia. Everywhere in the air-passages, as well as in the cavities of the mouth and pharynx, saliva, mucus, etc., readily collect if the patient is very ill. Expectoration is imperfect, and the constant dorsal decubitus favors the accumulation of secretion, especially in the lower lobes. The mouth and pharynx are harder to keep clean than under normal conditions. Fungi and bacteria collect in the secretion itself, as well as in the epithelium and particles of food which are left in the mouth, and these excite and keep up processes of decomposition. The inflammatory agents, which are carried into the air-passages with the inspired air, find everywhere favorable conditions for colonization and further development. From the upper portions they are drawn farther downward. From the larger bronchi the process invades the alveoli, and leads to catarrhal pneumonia. It is probably of significance, too, that in such very sick persons the vital resistance of the tissues has suffered, and that the development of such secondary inflammation is consequently facilitated. We must also bear in mind that many patients who are very ill have difficulty in swallowing. They get choked, and particles of food, with the germs of inflammation clinging to them, are carried into the

air-passages. These particles, which a healthy person could easily cough up again, remain there, are decomposed, and give rise to bronchitis and lobular pneumonia.

This is the explanation of the frequent development of lobular pneumonia in the course of diseases which are entirely dissimilar. We observe it especially in severe bedridden cases, in all patients with stupor, in severe infectious diseases, in meningitis, and also in cases of nervous disease, in which coughing and deglutition are impaired as a result of bulbar affections. In all such cases lobular pneumonia is to be considered a complication, and with reference to its origin deserves the name of inhalation pneumonia or deglutition pneumonia. We shall soon see that this form, under some circumstances, may pass into circumscribed gangrene.

Although in the preceding we have always spoken of "agents of inflammation" in general, the precise variety is not necessarily the same in all cases. In the lobular pneumonias occurring in the course of measles, whooping cough, influenza, etc., it is possible that the original specific germs may penetrate as far as the alveoli, and there cause an inflammatory exudation; but this is certainly not always the case, and there are many reasons why in these affections, as well as in all the others mentioned above, catarrhal pneumonia should be regarded as a secondary complication, dependent upon the secondary invasion of other varieties of agents which cause inflammation. Different microörganisms may probably act as such agents. So far as our present investigations go, streptococci seem most frequently to be the special cause of bronchopneumonia, and sometimes in other cases staphylococci, diplococci, etc. Clinically, we cannot at present make a complete separation on a strictly ætiological basis.

The development of lobular pneumonia from bronchitis is most frequent, as we know, in children and old people. The frequency of catarrhal pneumonia in childhood is due in part to the limited dimensions of the bronchi. Besides that, however, the diseases in which it is especially frequent—namely, measles and whooping cough—are children's diseases. In old people its comparatively easy development is due to imperfect expectoration, and perhaps to the diminished resistance of the tissues.

The mild cases of primary bronchitis scarcely ever lead to lobular pneumonia, but sometimes in children, and less often in adults, a severe febrile bronchitis may occasion the formation of pneumonic foci. In Erlangen, the author has seen a good many cases which cannot be regarded otherwise than as primary catarrhal pneumonia. We may also state that the inhalation of irritating chemicals may occasion lobular pneumonia as well as bronchitis.

Pathological Anatomy.—It is characteristic of catarrhal pneumonia that the inflammation is usually plainly circumscribed (*vide supra*), being limited to the territory of a small bronchus. Hence the name of "lobular" pneumonia or broncho-pneumonia, in distinction from croupous lobar pneumonia. An atelectasis (*vide supra*) of the affected lobule, arising from the plugging of the bronchus leading to it, often, but not always, precedes the inflammation. The atelectasis, of course, becomes pneumonia only in case germs enter the atelectatic spot. The inflammatory process itself consists in the exudation of a scanty fluid, which usually does not coagulate, and of numerous pus corpuscles (white blood corpuscles) into the lumen of the alveoli. With

this is usually associated more or less marked desquamation of the alveolar epithelium, in which we often find necrosis or fatty degeneration. The alveolar cavities are completely filled with leucocytes and epithelium; few if any red blood corpuscles are to be seen, although in some cases they may be more abundant. The vessels of the alveolar walls are hyperæmic, and the connective tissue also contains a few wandering leucocytes.

The inflamed lobules are readily apparent to the eye and the touch by their firm consistence, being devoid of air. Their color at first, from the blood contained in the inflamed part, is dark red ("splenization"), but later it becomes more grayish. A bit of such an inflamed spot, cut out with scissors, does not float in water but sinks, because it contains no air. The lobular boundary of the individual nodules is usually easily distinguished from the neighboring healthy tissue, but, by confluence of adjacent nodules, large portions of the lung, and even whole lobes, may become infiltrated throughout—generalized lobular pneumonia.

Symptoms.—1. *Primary Catarrhal Pneumonia of Adults.*—The primary catarrhal pneumonia which infrequently occurs in adults usually begins with the same phenomena as a severe attack of acute bronchitis. The patient feels prostrated, and has cough, dyspnœa, and pain on the side which is chiefly affected. There is almost never a pronounced initial chill, as in croupous pneumonia. The fever is usually not very high, from 101° to 103° F. (38.5° to 39.5° C.), but higher temperatures sometimes occur, especially at the beginning of the disease. The expectoration is simply catarrhal or mucopurulent, never mucosanguineous, as in croupous pneumonia. The physical examination gives almost invariably in one lower lobe many moist râles and a slight tympanitic or dull tympanitic percussion note. Marked dullness and definite bronchial breathing are rare. In the lower lobe on the other, unaffected side, we often find signs of a slight bronchitis; but in general the unilateral character of the symptoms is characteristic of primary catarrhal pneumonia, in distinction from ordinary bronchitis and secondary bronchopneumonia. In mild cases the fever lasts from four to eight days, but the disease may last two or three weeks or more. There is never a crisis; the fever ends gradually by lysis.

The ætiology of primary catarrhal pneumonia has been little studied. Many cases may be streptococcus pneumonia; we must also consider the influenza bacilli, since catarrhal pneumonia is especially common at the time of an influenza epidemic.

2. *Secondary Catarrhal Pneumonia.*—Most of the cases of catarrhal pneumonia develop, as we have already said, secondarily in the course of other affections, hence the symptoms are frequently overshadowed by those of the other diseases. There are often found at autopsy a few foci of lobular pneumonia in the lower lobes which gave rise to no clinical symptoms whatever.

In other cases, however, the development of extensive lobular pneumonia is of the greatest clinical significance. In many cases of chronic bronchitis, pulmonary emphysema, pulmonary tuberculosis, etc., in which the patient suddenly grows worse with a rise of temperature, the cause is certainly the development of foci of lobular pneumonia. Such complications may after a time completely disappear, or may cause a permanent aggravation of the original condition (e. g., tuberculosis). The onset of lobular pneumonia in

other acute diseases is very important. The disturbance of respiration forms the most striking symptom of the disease during the patient's life, and at the autopsy lobular pneumonia is shown to be the immediate cause of death. The largest part of the fatal cases of measles and whooping cough, and no very small part of those of diphtheria, scarlet fever, typhoid, smallpox, influenza, etc., are due, in the last instance, to the disturbance of respiration dependent upon lobular pneumonia.

Since a diffuse bronchitis, extending into the finer bronchi, almost always precedes the development of lobular pneumonia, and since it may also give rise in itself to marked disturbance in respiration, there is no sharp boundary to be drawn clinically between diffuse capillary bronchitis and lobular pneumonia. Only the experience, a hundred times repeated, that extensive capillary bronchitis is apt to lead to lobular pneumonia, permits us to suspect the latter, with considerable certainty, even if there is no direct clinical evidence of it. The most important objective physical sign of bronchopneumonia is the not very loud, medium moist râles, almost always to be heard in the lower lobes. These râles are often high-pitched. Dullness of the percussion note develops only when many lobular nodules coalesce and form a more extensive infiltration of the lung. Then we also find bronchial respiration.

Catarrhal pneumonia is almost always associated with fever, which is moderately high and generally has a remitting character. We must, of course, judge of the fever in connection with the primary disease and any other complications.

No general statements can be made as to the duration of secondary catarrhal pneumonia. Sometimes the acute symptoms last only a few days, in other cases they continue for weeks. Catarrhal pneumonia is not infrequently followed by pleuritic effusion, when the lobular infiltration reaches the pleura. Foci of lobular pneumonia may develop into abscess or gangrene, but this is rare.

3. *Catarrhal Pneumonia in Childhood.*—The catarrhal pneumonia of childhood is very characteristic and clinically important. It is observed most frequently in measles and whooping cough, and also in weak, atrophic, and rachitic children. The increased frequency of respiration is most striking. The breathing is superficial, but labored, as is shown by the contraction of the auxiliary muscles of inspiration and the play of the nostrils. We also notice inspiratory retraction of the lower lateral portions of the thorax as a result of the incomplete entrance of air. The number of respirations in a minute increases in children to sixty or eighty, or even more. In most cases the child has a frequent and apparently painful cough. Expectoration is entirely absent in small children. When it is present it shows no characteristic peculiarities different from ordinary catarrhal sputum. The general condition is always bad. The child is restless, apathetic, and more or less stupid. Its face is usually pale, but often quite cyanotic. The pulse is very rapid, and in small children may attain a frequency of 140 to 180 a minute. Fever is almost always present. It shows no typical course, it is now remitting and now intermitting, and toward evening it perhaps rises to 104° or 105° F. (39.5° to 40.5° C.). The occurrence of such a rise in temperature is not without value in the diagnosis of catarrhal pneumonia.

If in diffuse capillary bronchitis a high fever is present for some time, we may assume with considerable certainty that the lobular infiltration has already begun.

Physical examination furnishes direct evidence of the affection of the lungs, but its results are for the most part to be referred to the diffuse bronchitis and not to the lobular infiltration. Auscultation gives the most valuable signs. We hear over the lungs, in a greater or less extent, numerous fine and medium moist râles, often quite high-pitched. From these signs, strictly interpreted, we can diagnosticate merely bronchitis, but we may suspect pneumonia with the greatest probability. With very confluent broncho-pneumonia, auscultation sometimes gives bronchial breathing and bronchophony, besides the râles.

It goes without saying that little lobular foci, surrounded by normal lung tissue containing air, give no special signs on percussion. With numerous nodules running into one another, the percussion note is duller, and there is sometimes tympanitic resonance. The dullness is often first to be made out over a stripe extending along the vertebral column—so-called "stripe pneumonia." X-ray pictures bring out the lobular foci very distinctly.

An attack of extensive lobular pneumonia is usually quite protracted. Even in favorable cases the disease rarely lasts less than two or three weeks, and it may persist much longer. The course of the disease is apt to be irregular, relapses succeeding improvement. The chief danger of the disease lies in this tendency to a protracted course, extending over weeks and months. Many children finally die, not of the lobular pneumonia itself, but from the general weakness and emaciation following the tedious febrile disease. We must remember, however, that complete recovery may sometimes take place quite late in the disease.

The "transition of catarrhal pneumonia to caseation and tuberculosis" is a clinical fact with which physicians have long been conversant. In fact, we often find true tuberculous changes in the lungs of children who have died after a tedious illness, as a result of measles, whooping cough, etc. There can, of course, be no real question, however, of an actual transition from one disease to the other. In such cases either we have to do with an acquired tuberculous infection, which has found a favorable soil in an already diseased lung, or (what is probably more frequently the case) the disease of the lung has promoted the development of a previously existing tuberculosis. It is usually weak children, with a hereditary predisposition to tubercle, who succumb to tuberculosis as a result of the above-named diseases. The diagnosis of a developing tuberculosis is not always easy, since it is only rarely that marked phthisical changes—like dullness at the apex, cavities, etc., which can be made out by a physical examination—are found in the lungs. We can usually suspect tuberculosis only from the general conditions (emaciation, persistent hectic fever, hereditary predisposition, or some secondary tubercular disease, such as meningitis, etc.), especially as absolute proof, from the detection of tubercle bacilli in the sputum, is only rarely possible in children. [Sometimes the physician or a good nurse can obtain sputum on a stick wrapped with absorbent cotton, as the baby coughs it up before he has time to swallow it.]

Treatment.—Since we have already mentioned the proper treatment, in our description of the various diseases in which secondary pneumonia is especially prone to develop, we can now be brief. We have also laid repeated stress upon the possibility and the great practical importance of prophylaxis, which is self-evident from a just comprehension of the origin of lobular pneumonia. Besides keeping the nose, the mouth, and the pharynx as clean as possible, tepid baths, perhaps with cool douching, are the best means of preventing the development of lobular pneumonia, or of checking its further extension. Wet cold packs are often used with advantage (*vide infra*). It is an advantage, which is indeed to be considered in the second rank in comparison with the improvement in respiration, that by both the bath and the pack the febrile temperature is at the same time reduced.

In the treatment of the lobular pneumonia of children a wet pack including the whole body is the best remedy. A sheet is dipped in water, wrung out, and wrapped around the whole of the patient except his head and arms. Outside of this is to be placed a dry woolen blanket or a layer of oiled muslin. The temperature of the water employed should be 68° to 75.2° F. (20° to 24° C.). The higher the fever the colder should the water be, and the oftener, say every hour or two, must the pack be renewed. In milder cases and at night it may be allowed to remain for three or four hours. The beneficial influence of the pack is shown not only by the temperature, but still more by the respiration. It is often striking to see how much quieter the child becomes in the pack. If the breathing, despite this remedy, remains unsatisfactory, and the patient becomes more and more stuporous, the treatment must be changed to lukewarm baths of a temperature of 77° to 86° F. (25° to 30° C.), with douchings of colder water. It is sometimes advisable in severe cases to add to the water employed for bathing or for the wet pack a few handfuls of mustard. The stimulation thus exerted upon the skin is quite marked.

Among external applications to the chest, besides mustard plasters and poultices, dry cups are to be mentioned, which often do very good service in strong, older children, and especially in adults. We never need to use local blood lettings, however, in catarrhal pneumonia.

Of internal remedies, expectorants are most used. Chief among these are ipecac, apomorphin, senega, and benzoic acid. This last is particularly useful in the lobular pneumonia of children. In strong children the abundant collection of mucus in the bronchi may sometimes be relieved by the administration of an emetic, but we seldom need to resort to this. We should also be cautious in the use of narcotics. Stimulants (camphor, caffein) must be used in severe cases. Antipyretics may be given to reduce the temperature, but if cool packs be used, antipyretics can be wholly dispensed with. Inhalations or sprays are quite valueless in lobular pneumonia, yet it is recommended to keep the air in the sick-chamber constantly moist by hanging up wet towels, or by sprinkling with water. The room should also be as large and as well ventilated as possible. [Inhalations of oxygen may do much good.] The general hygienic treatment is of the greatest importance. One of the most important duties, of which the physician must always be mindful, is to keep up the patient's strength by sufficient and proper food. When convalescence sets in, complete restoration to health may be materially furthered by going to a suitable place in the country.

CHAPTER V

CROUPOUS PNEUMONIA

(Lung Fever. Lobar Pneumonia. Fibrinous Pneumonia. Pleuropneumonia)

Croupous pneumonia is a sharply defined febrile disease of the lungs, which, in the great majority of cases, displays a distinct individuality in its clinical, anatomical, and usually, also, its ætiological relations. Among the severe acute diseases it is decidedly one of the most important and frequent, and it is universally familiar even to the laity under the names of pneumonia or inflammation of the lungs. In most cases this disease appears quite suddenly, and often, apparently without any special cause, in persons up to that time in perfect health. Such cases are described as primary, genuine, or frank pneumonia. On the other hand, a case of croupous pneumonia will sometimes occur as a complication of all sorts of other diseased conditions (secondary pneumonia). The clinical picture of pneumonia in these latter cases, however, is usually obscure and not well characterized, and consequently the following description is especially applicable to the primary form of the disease.

Ætiology.—The thought that croupous pneumonia might be an acute infectious disease had impressed the majority of physicians for a considerable length of time, but this suspicion did not receive satisfactory substantiation until the more recent investigations in bacteriology had been made. For instance, Friedländer demonstrated a special form of bacillus in lungs affected with pneumonia. Then, later, A. Fränkel, and, soon after him, Weichselbaum, proved that while this "pneumonia bacillus of Friedländer" is to be regarded as the cause of croupous inflammation of the lungs in a few cases (just as we have a peculiarly malignant form of streptococcus pneumonia), yet in the overwhelming majority of instances the particular ætiological factor is the so-called *Diplococcus pneumoniæ* (pneumococcus).

The pneumonia diplococcus is characterized by its lancet-shaped outline ("like the flame of a candle"), and its frequent arrangement in pairs, the individuals of each pair having usually their broad ends apposed. Very often there are short chain forms. The pairs of diplococci are usually surrounded by a delicate capsule. This is especially easy to see in preparations of the sputum. We have not space to describe particulars as to the pure cultures of the pneumococci upon agar and similar media.

The pneumococcus is very virulent for mice, guinea pigs, and rabbits, and, when injected, causes a rapidly fatal pneumococcus sepsis.

Pneumonia diplococci are among the most widely distributed of pathogenic organisms. They produce severe disease not only in the lungs, but in many other organs. With regard to the development of the pneumonic infection, it is a particularly interesting fact that these diplococci have been not infrequently found in the mouths of healthy persons. This suggests the thought that the germs are drawn into the lungs with the inspired air, and there settle and proliferate when the conditions are especially favorable—for instance, if the resisting powers of the organism have been impaired, or perhaps if the diplococci are especially virulent. In animals the injection of diplo-

cocci into the lungs almost always excites pneumonia, but yet it is doubtful whether the incidence of the disease in man always bears out the surmise above mentioned. The severity of the constitutional disturbance at the very onset of many cases certainly justifies the supposition that at least often the infection takes place through the blood, and that the pathogenic organisms are carried to the lungs by way of the blood vessels. Diplococci are often to be found in the blood of pneumonia patients (*vide infra*). Certain clinical facts suggest that perhaps in individual cases there may be other modes of infection; for instance, by way of the intestine in cases with well-marked intestinal symptoms. Starting from the lungs, the diplococci may invade the pleura, less often the pericardium, the meninges, and other organs, so that they are invariably demonstrable in the pus of cases of metapneumonic empyema, pericarditis, and meningitis. From a clinical point of view their almost invariable presence in pneumonic expectoration (Fig. 45) is of the greatest importance. They can be easily demonstrated by the staining of a dried preparation with gentian-violet, although for absolute certainty of diagnosis further investigation is required.

Fig. 45. — Pneumonia diplococci from a pneumonic sputum.

With the infectious nature of pneumonia established, all the other alleged causes may, of course, be regarded as at most "predisposing causes." The old opinion, which is yet current, that pneumonia is due to catching cold, is to be received with great limitations, for croupous pneumonia is very frequently seen independently of any such influence. In many cases it will be found that an exposure to cold immediately preceded the commencement of the disease; but in these instances the cold is probably to be regarded merely as that circumstance which promoted the occurrence of the infection, possibly because of the resultant injury to the bronchial and pulmonary epithelium. This explains the fact that pneumonia is especially frequent in certain classes, for instance, among day laborers and soldiers. With regard to the so-called "traumatic pneumonia," the state of the case is similar to that of pneumonia due to cold. Patients from the laboring classes sometimes assert that they were taken ill as a result of heavy lifting or of a blow on the chest, but in such cases the subsequent stitch in the side was probably not the result of the injury, but a symptom of the disease which had previously begun to develop. But still, in some few cases it may be that a preceding trauma injures the pulmonary tissue in such a way as to promote the occurrence of the infection.

It is a strong argument in favor of our conception of pneumonia as an acute infectious disease that it may rarely be endemic. Extensive endemics of pneumonia, usually of quite a malignant character, have been repeatedly observed in single buildings, especially in barracks or prisons, as well as in tenement houses and other localities. It is possible that precisely these severe cases of endemic pneumonia are ætiologically different from the ordinary croupous form and occasioned by some other pathogenic organism; but it is also true that the diplococcus of pneumonia itself appears to vary greatly in its virulence.

Pneumonia does not, as a rule, show a decided epidemic character. In a

large population sporadic cases occur at any season. It has been observed, however, that the disease may become very frequent or almost completely disappear, and then again for a time be decidedly prevalent. In a limited way, therefore, it is quite proper to say that there are actual epidemics of pneumonia, and these, again, differ from one another in special peculiarities, particularly in their comparatively benign or malignant character. Most attacks occur in the winter or spring months, without any necessary relation, however, between the frequency of pneumonia and the occurrence of especially bad, wet, or cold weather.

Individual predisposition plays an unmistakable part in the disease, as we must suppose that it does in all infectious diseases. Like facial erysipelas and acute articular rheumatism, pneumonia is one of those diseases which is prone to attack the same individual several times. There are persons who have had acute pneumonia four or five or even more times.

We cannot affirm with certainty that the liability to pneumonia is due to a special bodily constitution. The strongest and most robust often fall ill with it, and, on the other hand, weak and delicate people, with a tendency to phthisis, are frequently attacked. Drunkards seem to have a special predisposition to the disease, but of course it is exceedingly hard to give any definite statistics upon this point.

Pneumonia occurs at any time of life, most frequently in youth or middle age; but it is by no means rare in early childhood, and also in more advanced years up to sixty or seventy. In general it is observed rather more often in men than in women.

[Defective house drainage seems to be a predisposing cause of pneumonia in some cases. A careful inspection of the local sanitary conditions is desirable, especially where more than one case occurs in a house.]

Pathological Anatomy.—The anatomical process in croupous pneumonia consists in the formation of a hemorrhagic, coagulable "fibrinous" or "croupous" exudation into the pulmonary alveoli and the smallest bronchi. The development of the exudation usually extends over one or more lobes to their whole extent, and, as the alveoli and finer bronchi are completely filled by the tough exudation, the spongy lung, filled with air, is changed to a firm tissue, devoid of air, except as it is penetrated by the large bronchi.

Since Laennec's day we distinguish three stages in the development of the process. In the first stage (stage of inflammatory engorgement, *engouement*) the lung is very hyperæmic, dark red, and the air contained in it is even now much diminished, but not entirely absent. The alveoli are filled with an abundant exudation, already hemorrhagic, but still fluid and not coagulated.

In the second stage (stage of red hepatization) the coagulation of the exudation is complete, and the lung has become throughout of the consistence of liver. The hepatized lung shows a somewhat increased volume, and is strikingly hard. The surface of the section has a red and manifestly granular appearance, which is due to the projection of the numerous little fibrinous plugs situated in the alveoli. With the knife we can scrape off a tenacious, creamy, grayish-red fluid from the surface of the section. In the small bronchi, divided by the knife, we find characteristic tubular bronchial casts.

In the third stage (stage of yellow or gray hepatization), which gradually develops from the second, the red surface of the section changes to a yellowish-

gray color, often mottled, while the lung grows anæmic and the exudation poor in red but rich in white blood corpuscles. The consistence of the lung is still dense but more friable. The fluid scraped from the surface of the section is more abundant, milky, and puriform.

The recovery from the process begins as the exudation becomes fluid. The fluid is in part absorbed and in part coughed up.

It is not necessary for every pneumonia to go through all three stages completely. In mild cases the process may stop sooner and recovery begin.

Concerning the finer histological processes in croupous pneumonia, the primary change is probably to be found in the injury and partial destruction of the epithelium in the alveoli and smallest bronchi, produced by inflammation due to the specific causes of the disease. As in every croupous inflammation of a mucous membrane (see the chapter on Diphtheria), a coagulable exudation is formed on the surface of the alveoli and smaller bronchi after the destruction of the epithelium. With the microscope we see the fibrinous network of the exudation filling the alveoli. Between its meshes lie numerous red blood corpuscles—red hepatization. Where there is any of the alveolar epithelium left, we often notice active proliferation—increase and growth of cells. Later on the white blood corpuscles increase, migrating from the vessels into the exudation—yellow hepatization. The red blood corpuscles are dissolved unless they are removed by expectoration.

The fibrinous exudation is also gradually dissolved, and this, as Fr. Müller has shown, results from chemical changes which are very similar in many respects to the digestive changes that albuminous substances undergo in the stomach and intestines. The coagulated albumen is broken up into soluble albuminoids and further decomposition products through the action of a ferment that is probably given off by the leucocytes. In addition to phosphoric acid, considerable quantities of the xanthin bases (xanthin and hypoxanthin) are formed from the disintegrated nuclei of the dying cells. All these processes facilitate the rapid absorption of the pneumonic exudation. The regeneration of the missing epithelium comes from the epithelium that has remained intact, and with that follows a gradual and complete *restitutio ad integrum*.

The whole process is comparatively brief, usually running its course in a week or ten days. The most frequent termination is in complete recovery. The other methods of termination, as well as the complications in other organs, will be spoken of in connection with the clinical symptoms. We may here mention simply that the pleura over the affected portion of the lung takes part in the inflammation, without exception, as soon as the disease reaches the periphery, and a fibrinous pleurisy, which is more or less intense, may then be recognized; hence the former use of the terms "pleuropneumonia" and "peripneumonia."

Croupous pneumonia usually spreads rapidly over a great part of the lung. It is very often quite sharply limited to a single lobe—"lobar pneumonia"—so that the septum of connective tissue between two lobes also forms a strict boundary between pneumonic infiltration and healthy lung tissue; but this boundary is by no means insurmountable, and quite frequently several lobes are wholly or in part attacked by pneumonia. According to all statistics, the lower lobes are more frequently affected than the upper. Isolated disease of the right middle lobe may occur, but it is much rarer than pneumonia of the

upper lobes. Of the two lungs, the right is attacked with decidedly greater frequency than the left. We have ourselves seen, in 244 cases, 137 on the right, 86 on the left, and 21 in which both lungs were attacked to a great extent. Simultaneous affection of the lower lobe on one side and the upper lobe on the other—quite a rare occurrence—is termed "crossed pneumonia."

General Course of the Disease.—In spite of the numerous modifications which the course of pneumonia may undergo in individual instances, we can still call pneumonia a typical disease, considering the great majority of cases. The subjective and objective symptoms dependent upon the local affection of the lung usually, but not always, take the chief place among the clinical phenomena. In this pneumonia differs from many other infectious diseases, such as typhoid, in which the local organic disease is subordinated to the general infection.

Pneumonia usually begins quite suddenly. In the majority of cases it starts with a pronounced chill of half an hour to an hour's duration, or at least with a marked and prolonged chilliness. The initial chill may attack the patient while in the best of health. Many patients are able to tell almost the very hour when, having been previously in perfect health, they were attacked by the disease. The chill comes on in the daytime, in the evening, or even in the middle of the night, after a previously quiet sleep. At the same time the patient almost always feels as if a severe illness were beginning. Almost at once he is obliged to give up work, has violent headache, and loss of appetite. Not infrequently there is a single initial act of vomiting. Sometimes there are at once pulmonary symptoms, such as a stitch in the side and cough. Usually, however, these phenomena do not develop until later (*vide infra*).

In other and somewhat rarer cases the beginning of pneumonia is more gradual. A prodromal stage of a few days, or even longer, precedes the severe illness. The symptoms are either of quite a general and indefinite nature, consisting of malaise, dullness, loss of appetite, and headache, or the prodromal symptoms point more strongly to a pulmonary affection. The patient complains several days, or even weeks, before the onset of the severe disease, of cough, thoracic discomfort, slight dyspnœa, and similar symptoms. At the same time it is not possible to determine certainly whether these prodromata are caused by an already existing pneumonia or not. It is undoubtedly true that in most cases we have merely a simple bronchitis which furnishes a favorable soil for the development of pneumonia; but perhaps the initiatory bronchitis may, in some cases, itself be an effect of the diplococcus infection, already begun, but not yet completely developed.

The subjective symptoms in the chest begin shortly after the onset of the disease, often on the very first day, but in other cases later. The patient has a stabbing pain in the side whenever he draws a deep breath; the respiration, therefore, becomes superficial and accelerated, and often somewhat irregular; his speech is interrupted by frequent pauses. In the further progress of a severe case the dyspnœa becomes extreme and the respirations very frequent. With the stitch in the side is associated a desire to cough. The cough is painful, and hence short, half suppressed, and quite frequent and troublesome. From the second day the expectoration may assume its characteristic viscid, rusty, hemorrhagic appearance. Physical examination gives on percussion and

auscultation the signs to be described more fully below. These are rarely to be found on the first day, but more frequently on the second, and sometimes not till later.

In well-marked cases the severe constitutional symptoms persist or grow worse. We observe general weakness, headache, complete loss of appetite, restlessness, stupor, and delirium. Herpes appears on the lips or nose; the bowels are sluggish, or, again, they may be loose; the urine is concentrated, and very often it contains a small amount of albumen.

Almost always the pneumonia is associated with high fever. The typical character of the disease and the peculiarities of the individual case are always well shown by the temperature chart. As the bodily temperature rises there is a corresponding increase in the frequency of the pulse.

The course varies greatly according to the previous individual circumstances, the severity of the disease, and the existence of complications. In the majority of cases, after a comparatively short duration, the disease takes a favorable turn. The beginning of improvement is often sudden, like the onset of the disease. After the symptoms have lasted for some five to seven days, or in rarer cases a shorter or a longer time, at a constant height or with increasing intensity, there occurs in the regular course of the disease a critical decline of the fever—frequently associated with quite a copious perspiration —and with that a very rapid improvement of all the other symptoms. In a short time complete recovery follows.

In other cases, however, the course is not so favorable. The disease may have a fatal termination. In a third small class of cases the disease finally takes a protracted course, which is usually due to the occurrence of sequelæ in the lungs or pleura.

To this brief sketch of the disease we must append a description of the special symptoms.

1. **Lung Symptoms.**—First among the subjective symptoms comes the characteristic painful feeling or "stitch" in the side. This probably always has its origin in the dry pleurisy which accompanies the pneumonia. It is therefore absent in the cases of central pneumonia (*vide infra*). In pneumonia of the lower and right middle lobes the pain is usually more severe than in pneumonia of the upper lobes. One result of the stitch in the side is the difficulty, or even the impossibility, of deep inspiration. Hence the patient's dyspnœa is considerably increased, and this explains the incongruity between the shortness of breath and the relatively slight extent of the pneumonia in many cases. If the pneumonic infiltration of the lung is extensive, of course the dyspnœa of the patient is also referable in part to the diminution of the respiratory surface. The subjective feeling of difficulty of breathing is prominent in the majority of cases, and it may become most distressing.

Cough is one of the most constant symptoms in pneumonia, and is usually very painful; hence the patient often tries to suppress it. Expectoration is apt to be very difficult at the onset of the disease, from the viscidity and scanty amount of the sputum; hence severe and distressing paroxysms of coughing are sometimes observed. The cause of the cough is probably not to be found in the affection of the alveoli, but in the coexisting bronchitis. The irritation of the pleura may also set up a reflex cough. In rare cases cough is entirely

absent in pneumonia. Except in the cases of limited or late localization (*vide infra*), we observe this absence of cough chiefly in the pneumonia of old or very weak people, and also, what is of practical importance, in the drunkard's pneumonia associated with delirium tremens.

The pneumonic expectoration is so characteristic that we can often make the diagnosis of croupous pneumonia from this alone. It consists of a very tough viscid mucus, which sticks fast to the bottom of the cup even when inverted, and contains an intimate admixture of blood, which gives it a more or less pronounced red or yellow hemorrhagic color. In individual cases there are numerous gradations. We usually call the pneumonic sputum "rusty," or "brick-red," or of a "prune-juice color," etc. Sometimes it has only a slight reddish or yellowish tint, and sometimes it consists almost entirely of blood. Often the sputum is very frothy. In some cases it assumes a peculiar grass-green ["greengage"] color, which is due to a change in the blood coloring matter, or to a mixture with bile pigment in "bilious pneumonia."

The red color of the sputum, as microscopic examination shows, is due to numerous red blood corpuscles, many of them still well preserved, mixed with it. They are, however, in part dissolved, and hence the uniform red color of the sputum. Separate spots containing much blood are often seen in it. Besides the red blood corpuscles, the microscope shows numerous partly swollen or fatty-degenerated pus corpuscles. We also see long threads of mucus; sometimes large, round, pigmented cells (alveolar epithelium?); and finally, in rare cases, ciliated epithelium and crystals of hematoidin.

FIG. 46.—Bronchial cast, ¾ natural size.

Fränkel's diplococci are, as we have said, almost always easily demonstrable in the expectoration, and of course numerous other bacteria besides.

We have still to mention the bronchial casts as important constituents of pneumonic sputum. Since they are usually rolled up together, we may not find them except by spreading out the sputum in water. They consist of the most beautiful casts of the small bronchi, with many dichotomous divisions, and are a product of the croupous inflammation extending into the bronchi. The casts of the smallest

bronchi are sometimes found in the form of "spirals," like those in asthmatic bronchitis (see page 224).

The amount of the pneumonic sputum is, as a rule, not very considerable, but it differs a good deal in different cases. The chemical examination of the sputum has so far given no remarkable results. The amount of common salt contained in it is quite considerable.

In many cases the pneumonic expectoration is absent, either because there is no cough or because the exudation is firmly coagulated and is absorbed without ever liquefying. Sometimes it is very tough and slimy, but without any admixture of blood; in other cases the sputum is simply catarrhal, when present at all, and then, of course, it comes not from the parts infiltrated with pneumonia, but from the catarrh of the larger bronchi. In many severe cases the hemorrhagic expectoration soon assumes more of a purulent character. We often find simple catarrhal sputum, too, besides the characteristic pneumonic sputum.

The pneumonic sputum is sometimes seen in the first or second day of pneumonia, but it may not appear until later. With the beginning of resolution it gradually loses its characteristic appearance. The expectoration then becomes less tenacious and simply mucopurulent, and finally disappears entirely.

Physical Examination.—Inspection shows no especial anomaly in the general contour of the thorax. A marked bulging of the affected side occurs only when there is also abundant effusion into the pleural cavity. The action of the thorax in respiration is very important. Even with a limited pneumonia we often notice a very marked delay and limitation of motion of the affected side on inspiration. This is due in part to the pain in the side, which comes on with every deep inspiration, and also, in extensive pneumonia, of course, to the physical conditions resulting from the anatomical changes. The unaffected portions of the lung act all the more forcibly.

The acceleration of respiration is very striking, its frequency increasing to thirty or forty, or even more, a minute. We have repeatedly counted sixty respirations in adults, even in cases that finally resulted favorably. The breathing is shallow, but yet, in all severe cases, labored, and often also irregular, as a result of pleuritic pain or cough. We see the inspiratory contraction of the sterno-cleido-mastoids and scaleni in the neck, and often in the face a marked dilatation of the nostrils on inspiration. If there is marked dyspnœa the patient sometimes reclines in bed with the upper half of the body raised. The cheeks and lips are cyanotic. There is often a sharp contrast between the pale portions of the face near the corners of the mouth and the striking feverish and slightly cyanotic flush of the cheeks.

The results of percussion are directly dependent upon the changed physical condition in the lung, due to the anatomical processes. In the beginning of pneumonia, so long as the total amount of air in the lung remains but little altered, the percussion note is clear, but when the elasticity and tension of the tissue in the diseased portion of the lung diminish, the resonance often becomes quite tympanitic. With increased exudation into the alveoli and smallest bronchi the amount of air in the lung constantly grows less, and therefore the percussion resonance becomes very dull, but it usually retains its tympanitic timbre. Since the pneumonic lung is rarely absolutely deprived of air

—for a certain amount is always left in the larger bronchi—the percussion resonance seldom becomes so completely dull or flat, as it does, for example, with a large pleuritic effusion. The sensation of resistance upon the percussion of a pneumonic lung is likewise much less marked than over a pleuritic exudation. A marked sense of resistance implies, therefore, an unusual degree of involvement of the pleura in the inflammation. As soon as the absorption of the exudation begins, the volume of air in the lung increases, and the percussion note becomes clearer, but remains for some time still markedly tympanitic, until the lung has regained its normal tension and elasticity. We have also to note that the intensity of the dullness in croupous pneumonia is sometimes subject to quite marked variations, since the secretion retained in the bronchi is at one time abundant and at another, after expectoration, scanty.

The extent of the dullness or of the tympanitic resonance is naturally dependent upon the extent of the anatomical process. Small and central infiltrations may entirely escape detection by percussion.

Auscultation is of almost greater importance than percussion in the detection of a beginning or limited pneumonic infiltration. The auscultatory signs depend upon the presence of the pneumonic exudation, and upon the consequent transformation of the lung into a firm tissue devoid of air except in the larger bronchi. In the beginning of the disease we hear over the affected portions coarse or fine râles, and very often, too, the characteristic crepitant râle on inspiration discovered by Laennec. This arises because the walls of the alveoli and smallest bronchi, which are glued together by the viscid exudation, are torn apart at each inspiration. The crepitation, however, is neither pathognomonic of pneumonia, nor heard in every case of pneumonia. With increasing infiltration, bronchial breathing replaces the vesicular. The bronchial breathing in pneumonia is usually very loud, sharp, and close to the ear. When the infiltration is very complete there is often to be heard a pure and loud bronchial respiration, without any adventitious sounds; but of course there are often heard, besides the bronchial breathing, high-pitched, moist râles in greater or less abundance. It is especially true that with the commencement of "resolution"—that is, as soon as the exudation becomes more fluid, abundant moist râles reappear, enough to obscure more or less the bronchial respiration. These râles are, for the most part, rather coarse, moist, and have a musical character. Often we hear at this time the characteristic crepitant râle again (*crepitus redux*). The râles gradually disappear, the respiratory murmur loses its bronchial character, becomes harsh and indefinite, and finally is normally vesicular once more.

We often hear a few rhonchi over the unaffected portions of the lungs. On the diseased side, the respiratory murmur is often more or less diminished because of the diminished respiratory movement. Otherwise respiration in the unaffected portions of the lungs is usually completely normal.

The auscultatory signs just described undergo an important change if the larger bronchi leading to the affected portion of the lung are completely plugged by the secretion, as they are quite liable to be. The respiratory murmur may then almost entirely disappear, and we hear, perhaps, only here and there a few obscure râles. Since such a plugging may be very transitory, we understand why in one day, over the same portion of the lung, we hear first

loud bronchial breathing and râles, and then quite obscure and diminished breathing.

Wherever there is bronchial breathing, we hear marked bronchophony. Sometimes one can discover a beginning pneumonic infiltration by bronchophony sooner than by any other physical sign. The vocal fremitus persists or is somewhat increased over a pneumonic lung so long as the large bronchi are open; but when they become plugged, as they are quite liable to be, the vocal fremitus is weakened or wholly abolished; and again, the voice sounds are weakened whenever there is a considerable amount of pleurisy accompanying the pneumonia.

We have yet to add a few remarks about the parts of the lung in which we may expect first to perceive the physical signs of pneumonia, especially the auscultatory signs.

In the first place, we should never neglect to examine carefully the lateral portions of the thorax and the axillary region when we suspect a developing pneumonia. We often find the first râles here in pneumonia of the lower lobes. The first signs of infiltration may be found in the posterior middle portion of the thorax—that is, in the upper part of the lower pulmonary lobes—and thence they extend downward. Pneumonia of the upper lobes begins just as frequently behind in the apices as in front in the infraclavicular fossæ. Isolated pneumonia of the right middle lobe also occurs, to be made out in front, on the right, between the fourth and sixth ribs. This may remain confined to the middle lobe, or extend to the neighboring lobes.

Few general statements can be made about the mode or the rapidity of the extension of pneumonia, since in these respects the greatest differences are observed. The infiltration may remain confined to a small portion of the lung, or again it may spread over a whole lobe or more in a short time, even in one or two days. We call the pneumonia whose constant extension by contiguity we can follow from day to day wandering pneumonia (*pneumonia migrans*), or, from a purely superficial resemblance, which has given rise to many wrong ideas, "erysipelatous pneumonia." In these cases all the signs of resolution are present in the parts first attacked, while the parts affected later are found still at the height of the disease, or in the beginning of infiltration; but we may also find in the autopsies of wandering pneumonia the parts of the lung affected later in a more advanced stage (gray hepatization) than the parts first attacked, which are still in the stage of red hepatization—that is, the inflammatory process in such cases seems to go through a more rapid evolution in the portions of lung later affected. Wandering pneumonia is almost always severe and quite protracted.

Pneumonia in rare cases progresses by leaps. Such cases have been termed erratic pneumonia. In severe cases it is not unusual for both lungs to be affected. We then find the pneumonia either in both lower lobes, or in the lower lobe on one side and the upper lobe on the other side.

2. **Pleura Symptoms.**—As we have already mentioned, every case of pneumonia which reaches to the surface of the lung is associated with a fibrinous pleurisy, and, furthermore, it is not impossible that there should be an infection of the pleura without any direct extension from the neighboring pulmonary tissue. In many instances, the mild pleurisy which attends a pneumonia causes no physical signs. On the other hand, the pain in the side in pneu-

monia is probably always referable to the involvement of the pleura. In other cases the dry pleurisy attracts attention by the clearly audible friction, which may often be very loud, and is sometimes appreciable to the touch, if the hand is laid upon the side. We rarely hear the pleuritic friction sound in the beginning of pneumonia, but more frequently in the later stages, and perhaps not till many days after the crisis has taken place.

The cases in which pleurisy with effusion complicates pneumonia are more important. This may occur quite early. The abnormality of the clinical course is shown in these cases, as a rule, by the irregular behavior of the fever. There will be no typical crisis, but instead a slow lysis; or after the crisis occurs, the temperature will rise again. In most instances the exudation is serous, but it may be purulent (metapneumonic empyema). Long-continued fever should arouse suspicion of an empyema. In the pus of such cases of empyema the pneumonic diplococcus above mentioned (see page 249) has been repeatedly found. In two fatal cases the author has seen a hemorrhagic pleurisy with a large amount of coagulated blood in the pleural cavity.

The diagnosis of pleurisy with effusion complicating pneumonia is seldom difficult. The percussion resonance is duller, and the sense of resistance is more marked than in pure pneumonia (*vide supra*). The respiratory murmur and the vocal fremitus are constantly diminished and finally entirely absent. The symptoms of pressure on the neighboring organs and cavities, the heart, the liver, and the semilunar space (see page 350), are especially important because they are most unequivocal. An exploratory puncture with a Pravaz's [hypodermic] syringe, that has been carefully cleansed and disinfected, gives a certain and safe method of recognizing pleurisy in doubtful cases. When there is suspicion that an empyema has begun, an exploratory puncture is imperative.

A moderate degree of pleurisy may somewhat delay the course of the disease, but it has no special significance. Large effusions, however, may decidedly increase the difficulty in respiration and the duration of the illness. Again, the pneumonia may recover, leaving the pleuritic effusion quite undisturbed. In pneumonia of an upper lobe, too, the pleurisy may develop below and lead to an effusion there, while the lower lobe itself remains quite free from pneumonia. Metapneumonic empyema invariably requires surgical intervention, but after operation it almost always pursues a rapid course to recovery.

3. **Circulatory Apparatus. Blood.**—The pulse is accelerated from the beginning of the disease. In cases of moderate severity its frequency reaches 100 or 120; and, in very severe cases, a still higher rate up to 140 or 160 is seen, and is always a dangerous symptom. This high rate of the pulse does not have as bad a significance in children as it does in adults. The consideration of the quality of the pulse is important. Smallness, weakness, and irregularity of the pulse are of bad omen as symptoms of the onset of cardiac weakness. The attacks of collapse, which sometimes come on quite suddenly in severe cases of pneumonia as in other acute diseases, are especially dangerous. They occasion sudden weakness of the heart with a very small and frequent pulse. The temperature sinks to subnormal, 95° to 93° F. (35° to 34° C.). The peripheral parts, the nose and extremities, become cool, pale, and somewhat cyanotic. The general weakness and prostration become extreme. The collapse may be recovered from, especially with timely assistance, but patients may die in it.

Pericarditis with fibrinous or serofibrinous exudation is one of the most serious cardiac complications. This can always be explained by a direct conduction of the inflammatory process from the neighboring pleura, and is therefore somewhat more frequent in left-sided pneumonia than in right. It is a serious matter. Its diagnosis is seldom difficult if we make a careful physical examination of the heart, but with very severe and extensive symptoms in the lungs a complicating pericarditis may be overlooked.

A slight fresh endocarditis is sometimes found at the autopsy, but it has no clinical significance. Diseases of the cardiac muscle, especially fatty and parenchymatous degeneration, may be discovered *postmortem,* but they are by no means frequent. In very weak persons, drunkards, etc., who die of pneumonia, we sometimes, indeed, find the heart remarkably flabby, with the right ventricle dilated, but in many cases of pneumonia we find the muscle of the heart at the autopsy perfectly normal. Here we almost always have to do with conditions of the heart which existed previously to the pneumonia, and merely became prominent during its course. When persons of vigorous and healthy constitution die of pneumonia, as, indeed, seldom occurs, the myocardium is found at the autopsy essentially healthy. It must be emphatically stated that our present knowledge does not enable us to establish before death any positive relation between the histological condition of the cardiac muscle and its functional ability. Frequent experience has made us certain of this fact.

With regard to the blood, there is usually a well-marked leucocytosis in pneumonia. The exact count shows, not infrequently, twenty to twenty-five thousand or more of leucocytes in a cubic millimeter. With the crisis of the fever the number of leucocytes also falls abruptly, while in case of a pseudocrisis their number remains high. After the crisis, the lymphocytes, whose number is greatly diminished during the course of the disease, show a decided increase ("post-infectious lymphocytosis"). In some instances the leucocytosis is not marked. This is particularly frequent in severe and fatal cases, so that the absence of leucocytosis is regarded with some justice as an unfavorable element in prognosis. Pneumococci can often be found in the blood. Their presence in large numbers indicates a severe infection. Accordingly, we find a bacteriæmia more especially in cases that have no marked leucocytosis.

4. **Digestive Apparatus.**—In severe cases of pneumonia the tongue is dry, coated, and quite like the tongue in typhoid. The appetite is also almost wholly lost from the beginning. Vomiting is not infrequent, especially in the beginning of pneumonia, and it also occurs later. It is observed with especial frequency in the pneumonia of children. Severe symptoms on the part of the intestinal canal are rare. As a rule, the bowels are constipated, but there are also cases in which diarrhea is so troublesome that we must believe that the mucous membrane of the intestine is considerably involved in the morbid process (*vide infra*).

The complication of pneumonia with jaundice has a certain significance, but its causes are not always very clear. It is apparently sometimes due to an accompanying catarrh of the duodenum. In other cases the veins of the liver, dilated from stasis, may exert a pressure on the bile ducts. Slight jaundice has no special significance, and is frequent, even in mild cases; a marked jaundice, however, is seen only in severe cases, especially in drunkard's pneumonia. We call such cases, associated with jaundice, "bilious

pneumonia." They have often other severe gastro-intestinal symptoms, such as vomiting, diarrhea, and meteorism, and severe nervous symptoms, such as stupor and delirium.

The liver may be somewhat enlarged, usually because of passive congestion. The spleen is often moderately swollen, particularly in severe cases, just as in other infectious diseases (acute splenic tumor).

5. **Kidneys and Urine.**—The infectious character of pneumonia is also shown by frequent involvement of the kidneys. Careful examination of the urine almost always shows a trace, or even a considerable amount, of albumen. However, this is very seldom of serious import, and vanishes promptly after the crisis. In many instances the changes in the urine are so considerable as to show an acute nephritis. There is a large amount of albumen, with casts, epithelium, and blood, in the urine. But even these cases of genuine pneumonic nephritis, which usually develop about the third to the sixth day of the illness, seldom prove to be serious, and scarcely ever result in œdema, uræmia, or other complications. Usually they get well rapidly. In one single case the author has observed a transition into chronic nephritis. The way in which pneumonia causes albuminuria and nephritis—between which no sharp dividing line can be drawn—is probably by the production and excretion of toxins.

Great weight was formerly laid upon the diminution of the chlorids in the urine of pneumonia. In fact, the precipitate of chlorid of silver, when we put a drop of solution of nitrate of silver into the urine, may be very slight or entirely absent. The chief cause of this diminution of the chlorids is the small amount of nourishment taken by the patient, but we must also bear in mind the large amount of chlorid of sodium contained in the pneumonic exudation, and the retention of the chlorids in the body.

Great significance was also formerly ascribed to the abundant sediment of sodium urate (exceptionally, uric acid) which is often noticed on the day of the crisis. This is known as brick-dust sediment (*sedimentum lateritium*), and is perhaps due in part to a material increase in uric acid from the breaking down of the increased number of leucocytes in the blood, or it may be derived from the degenerated nuclei of the cellular pneumonic exudate. On the other hand, it should be borne in mind that the conditions for the deposition of sediment are especially favorable on the day of the crisis. The urine is scanty in amount because perspiration is so excessive, and hence it is concentrated and relatively very acid. It is, therefore, natural for the urates contained in it to be deposited in the form of a sediment.

Pneumonia, in common with most of the other acute febrile diseases, is attended with an increased secretion of urea during the disease. The greatly increased excretion of urea in the days following the crisis is due in great part to the absorption of the pneumonic exudate (*vide supra,* page 252). The fact is of theoretic interest that during the resolution of pneumonia the urine often contains a demonstrable quantity of peptone, which is, in all probability, due to the destruction of the cells in the pneumonic exudation and their absorption into the blood.

6. **Nervous System.**—As in every severe febrile disease, nervous symptoms of a mild type are very rarely absent in pneumonia. Among the nervous symptoms are general weakness and dullness, and especially headache, which

is often very intense, and is usually increased by coughing. The onset of more severe cerebral symptoms, particularly delirium, is of great importance. Delirium may appear in any case of severe pneumonia, but it is most marked and has peculiar characteristics in alcoholic subjects. This delirium gives the pneumonia of drunkards (*vide infra*) its characteristic stamp.

The usual cerebral symptoms in pneumonia do not correspond with macroscopic changes in the brain, but result from the poisoning of the body with the toxins of the pneumonia diplococcus; and yet there is also a true cerebral disease which has beyond a doubt a special relation to pneumonia, although it is an infrequent complication. We refer to purulent cerebro-spinal meningitis. This complication is particularly apt to appear at times when an epidemic of cerebro-spinal meningitis prevails, but it has been repeatedly observed at other times. The diagnosis of pneumonic meningitis may be obscured by the severe constitutional disturbances. Factors of importance are: the stiffness of the back and the neck; the pain in the head and in the nape of the neck; the stupor, changing to deep coma; and, in many cases, optic neuritis, demonstrable by the ophthalmoscope. The termination of a well-marked case of meningitis is probably invariably fatal, but we may have milder meningeal symptoms in pneumonia, such as pain and stiffness in the neck, followed by complete recovery. With regard to the development of this meningitis, it is probably to be regarded as a true metastatic inflammation, inasmuch as the pneumonia diplococci have been repeatedly found in the pus of the meningitis. As to the path which the pathogenic germs take to reach the meninges, we are not yet certain, but suppose that they travel along the lymph channels of the intercostal nerves into the meninges surrounding the spinal cord, and thence farther to the membranes of the brain.

7. **Skin.**—The frequent appearance of herpes in the course of pneumonia is characteristic, and is of diagnostic importance. It appears from the second to the fourth day of the disease, or sometimes later. Its ordinary seat is on the lips, especially at the corners of the mouth, also on the alæ of the nose, and more rarely on the cheeks or the ear (*herpes labialis*, *nasalis*, etc.). It has been seen only very rarely on other portions of the body besides the face, for example, on the forearm and the buttock, and in some cases on the cornea and on the mucous membrane of the tongue or gums. The herpes does not always come out all at once, but in fresh crops on successive days. We have several times seen two eruptions of herpes separated by an interval of several days. In repeated instances, under our own observation, herpes labialis, with a fresh rise of temperature, appeared some days after the crisis had taken place. Herpes may be extensive in the mildest cases, while it is particularly apt to be scanty or absent in severe cases. We are, therefore, on the whole, inclined to regard a well-marked eruption of herpes as of favorable prognosis. The true cause of the herpetic eruption is unknown. One might think of referring it to the action of toxins, just as in the herpes of other infectious diseases, such as intermittent and relapsing fevers, and epidemic meningitis. Other affections of the skin are of rare occurrenec. We have seen urticaria in some cases. The jaundice occurring in pneumonia has already been described.

8. **Course of the Fever** (see Figs. 47 and 48).—Pneumonia is, almost without exception, accompanied by a more or less high fever with a very typical

course. In the beginning of the fever the temperature rises very rapidly to a high point. Even during the initial chill the bodily heat increases from normal to about 104° F. (40° C.) and over. There are at present no observations to show whether, in the cases of pneumonia that begin gradually, there is also a gradual increase of the fever. During the course of the disease the fever shows on the whole a continuous or remitting character, but there is with this a decided tendency to single deep falls of temperature. Since these at first may easily be taken for the actual occurrence of crises, although later they are proved by the renewed rise in temperature to be a mere temporary decline in the bodily heat, they are termed pseudo-crises. Pseudo-crises are usually seen in the first days of the disease, but in some cases they appear later and, what is remarkable, they are more apt to appear on those days, such as the fifth or seventh, on which the true crisis is apt to occur. They may be repeated one or more times, giving the fever a decidedly intermitting character. These intermitting pneumonias, so called from the course of the fever, have nothing at all to do with malaria, which fact must be especially noted because of the frequency of erroneous statements.

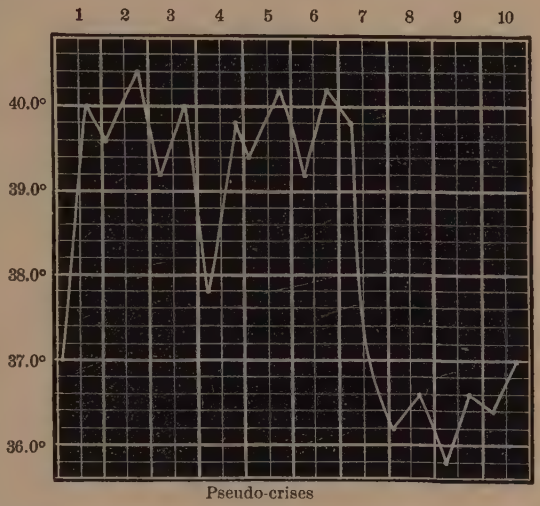

FIG. 47.—Example of the temperature-curve in croupous pneumonia. (Personal observation.)

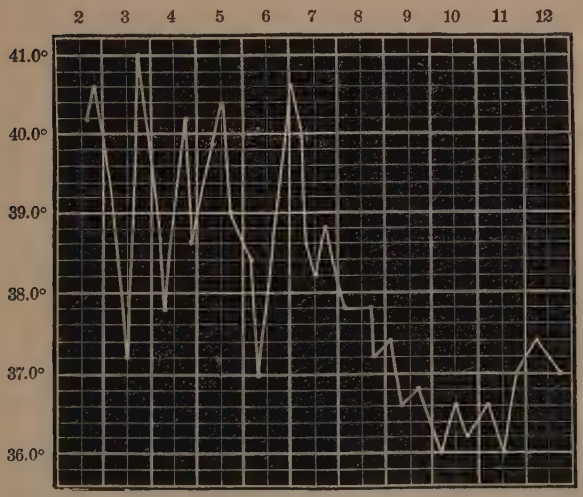

FIG. 48.—Example of the temperature-curve in "intermitting pneumonia." (Personal observation.)

The fever may be decidedly high in pneumonia, often reaching 104° or 106° F. (40° to 41° C.). The highest temperature observed by us was 107.8° F. (42.1° C.). This was temporary. There is a certain parallelism between the height of the fever and its severity to this extent, that severe cases are often associated with persistent and especially high fever. But sometimes the most severe and even fatal cases have a comparatively low temperature—between 101° and 103° F. (38.5° to

39.5° C.). The highest temperatures are especially common in the first days of the illness, and yet, in spite of very high fever at the onset, the general course of the disease may turn out to be favorable, for the crisis may occur on the second or third day (*vide infra,* rudimentary and abortive pneumonia). In severe cases the progress of the disease often stamps itself plainly on the temperature. The pseudo-crises correspond to temporary improvements, and the fresh exacerbations of temperature to the invasion of a fresh lobe of the lung. We have certainly not seen a special rise immediately before the crisis—the so-called *perturbatio critica*—so often as many statements would lead us to expect. We have seen a gradual decline in temperature quite frequently in the closing days in fatal cases, but the opposite condition also obtains. A marked rise before death is not peculiar to pneumonia, but it does occur when there is a complicating meningitis.

The decline of the fever is the most characteristic portion of the pneumonia curve. The fall in temperature usually comes on in the form of a decided crisis. Generally in the night there is a sinking of the temperature with a more or less abundant perspiration, in which, as a rule, the temperature may reach a subnormal point—96° to 95° F. (36° to 35° C.). The critical decline is often broken by new and slight elevations of temperature, so that on the morning of the next day there may be a definite increase of fever, the so-called protracted crisis. Only in a comparatively small number of cases does the fever end by lysis, in which the temperature goes down like steps. The duration of lysis is seldom more than three or four days at most. A decline of temperature by lysis is most frequent in severe and protracted cases, in so-called typhoid pneumonia (*vide infra*), and also particularly in *pneumonia migrans.*

After the final crisis has occurred, the active pneumonic process ceases. The day of the crisis is therefore reckoned as the last day of actual illness. The pneumonia makes no advance after that, but resolution and absorption of the exudation and the restoration of the patient's strength still take time. Hippocrates knew when the time of the crisis occurs, and that the odd days, especially the fifth and the seventh, have a special significance in regard to it. In an infectious disease that has a typical course there can be nothing strange in the fact that the cessation of fever, to a certain degree, is associated with a definite period of time; but Hippocrates' rule has frequent exceptions. The crisis sometimes occurs on the ninth, the twelfth, or the thirteenth day, and even later, and, on the other hand, there are quite short pneumonias of but one or two days' duration (*vide infra*).

In the days following the crisis the temperature, which, as we have said, falls to subnormal, regains its normal height. The pulse, which usually sinks to fifty or sixty during the crisis, when it often shows a slight irregularity, reaches its normal frequency again in a few days. We are quite apt to see, in the days immediately following the crisis, a slight temporary increase of temperature again, 100° to 102° F. at most (38° to 39° C.), but this has no special significance.

In cases which run their course regularly, the signs in the lungs upon auscultation and percussion become normal again in about six or eight days after the crisis. Often the time is even shorter than this, or it may be longer. Abnormally delayed resolution will be mentioned below.

Special Peculiarities and Anomalies in the Course of Pneumonia.—1. *Pneumonia in Children.*—Besides the common lobular pneumonia there is also a genuine, lobar, croupous pneumonia in children, which is by no means so rare as some authors formerly supposed. An initial chill is seen only in older children; initial vomiting, however, is very common in children. In many cases severe cerebral symptoms, like convulsions, drowsiness, or delirium, obscure the pulmonary symptoms at first. The further course, the development of physical signs, the fever, and the complications, are quite analogous to the appearances in adults. The pneumonic sputum is only exceptionally obtained for observation in children under eight years of age. In previously healthy children the prognosis of croupous pneumonia is almost invariably favorable.

2. *Pneumonia in old people* is, on the other hand, always dangerous. It may begin suddenly, as in people of middle age, but often it begins more slowly and insidiously. Its course is marked by the speedy onset of great weakness and debility. Nervous symptoms, like delirium, are not infrequent. Often there is fatal weakness of the heart.

3. *Drunkard's Pneumonia.*—We see croupous pneumonia in drunkards with remarkable frequency. The usually severe and dangerous course of the disease is due to the feeble resisting powers of their impaired organs. It is characterized by delirium tremens, which usually develops in the first days of the disease. The patient's mind is disturbed, he is very restless, constantly tries to get out of bed, and he fumbles night and day with his bedclothes or nightgown. The alcoholic character of the delirium is shown by the patient's whole manner, the tremor of the hands and tongue, and the cast of his thoughts, which are usually happy but exceptionally anxious and terrified. His mind wanders to his former occupation or his usual boon companions, and the like. In some cases the patients are noisy and very violent, strike out, destroy surrounding objects, etc. Under these circumstances they often believe themselves involved in tavern brawls. The alcoholic delirium is almost always associated with hallucinations. The hallucinations of little moving black figures are especially characteristic. They are either animals, rats or beetles, or little black men and similar weird shapes, and they give him much trouble. The subjective symptoms of pneumonia are wholly in the background. No delirious patient with pneumonia complains of cough, pain in the chest, or dyspnœa. Careful objective examination is the only thing that confirms the diagnosis. Very often patients with a happy delirium serve to entertain those about them, until suddenly very severe symptoms arise, and they become somnolent and succumb, with the symptoms of pulmonary œdema. The prognosis of drunkard's pneumonia, therefore, is always to be regarded as very unfavorable.

4. *Pneumonia in Preëxisting Chronic Diseases.*—Croupous pneumonia is occasionally seen in all forms of chronic disease. It is especially dangerous in persons who are already enfeebled, or afflicted with chronic cardiac or pulmonary disease, such as phthisis or emphysema. The pneumonia which often attacks patients with emphysema is clinically important, since emphysema may render the objective evidence of pneumonia very obscure. The croupous exudation does not completely fill the dilated alveoli; hence decided dullness and bronchial breathing are absent.

5. *Pneumonia with Late Localization—Central Pneumonia.*—Cases are quite often seen whose beginning, course, and subjective symptoms correspond throughout to a croupous pneumonia, but in which the objective evidence of pneumonic infiltration evades the most careful examination. The disease begins with a chill, the fever is high, the patient complains of pain in the chest, which is usually slight, there is, perhaps, herpes, but not till the fourth, fifth, or sixth day can we make out anywhere any bronchial breathing or crepitant râles. In other cases even the crisis may set in before we are able to localize the pneumonia with certainty. In most of these cases we probably have to do less with an actual late localization than with a central infiltration which nowhere approaches the periphery, and hence is made out objectively only late or not at all. A careful examination of the sputum is of the greatest diagnostic importance, since it sometimes has a perfectly characteristic appearance in spite of the absence or the indefinite character of the physical signs. If there is no sputum, the diagnosis may, of course, remain very uncertain. The appearance of herpes and a critical fall in the temperature make the diagnosis of a pneumonic infection probable even in these cases. In one such case the author observed the development of a pleuritic friction rub upon the first day after the crisis, confirming the diagnosis of pneumonia after the disease had run its course. An X-ray examination is of great value in such cases.

6. *Rudimentary and Abortive Forms of Pneumonia—Unusual Localizations of the Pneumonic Infection.*—Particularly at times of a pneumonic epidemic, but also at other times, the author has observed illnesses of short duration but often with high temperature which did not seem like clear cases of pneumonia, although they still were, in all probability, to be regarded as pneumonic—i. e., due to infection with the specific diplococci. Cases of this sort usually begin suddenly with a chill, headache, and high temperature. Generally there is a cough and pain in the chest. Sometimes, however, there are no thoracic symptoms at all. The physician expects pneumonia to develop, but instead, on the first, second, or third day, the fever ceases abruptly, no changes in the lung having been discovered. Very often there will be a *herpes facialis* in such cases, and we doubt not that many instances of so-called *herpes febrilis,* or *febris herpetica,* are really cases of pneumonic infection, without any other localization than the herpes. In other instances, upon careful examination there will be found, at some place in the lungs, a slight crepitation, or limited bronchial breathing; but the process does not extend; and in a very brief time, after a day or two, the fever ceases (rudimentary pneumonia; abortive pneumonia).

In this connection we may remark that the diplococcus infection may be localized in still other places. Thus, for example, we regard it as very probable that many cases of sore throat or acute enteritis, associated with the sudden onset of high fever and with herpes, and especially when seen at the time of an epidemic of pneumonia, are due to diplococcus infection. Associated with these attacks there may also be a late development of pulmonary symptoms.

7. *Typhoid Pneumonia—Asthenic Pneumonia.*—By typhoid pneumonia we mean those cases in which, besides the local pulmonary symptoms, which may be either slight or well marked, there are remarkably severe general

symptoms. The cases do not often begin as suddenly as ordinary pneumonia, but more gradually, like typhoid. Even at first the general symptoms, such as great dullness, loss of appetite, or headache, predominate over the thoracic symptoms. At the height of the disease there is a decided typhoidal state, stupor, delirium, a very dry tongue, great general weakness, and also enlargement of the spleen, and frequently mild jaundice, albuminuria, etc. Such cases are to be regarded as pneumonia with an unusually severe general infection (or intoxication). They sometimes occur in epidemics. It is said that pneumonia of the upper lobes shows a somewhat more frequent tendency to severe nervous symptoms than pneumonia of the lower lobes. Recovery from this typhoid or asthenic pneumonia, which may last two weeks or more, often follows by lysis. Typhoid pneumonia is by no means a sharply defined disease. The term serves merely as a short name for the grave constitutional disturbance. Clinically it is impossible to distinguish it sharply from *pneumonia migrans*, bilious pneumonia, and other forms. We must await further bacteriological investigations to determine whether there may not be some other special pathogenic organism in many cases of severe character.

Some especially severe forms of pneumonia, of apparently epidemic or contagious nature, have been recognized as streptococcus pneumonias. In this connection may be briefly mentioned, also, the peculiar and severe pneumonia which is contracted from diseased parrots (so-called psittacosis). On the other hand, it is not improbable that another important factor may be a variation in the virulence of the ordinary pneumococcus as well as the greater or lesser invasion of the blood by the pneumococcus.

Pneumo-typhoid is to be carefully distinguished from typhoid pneumonia, although from a clinical point of view the diagnosis between them is often no easy matter. By pneumo-typhoid is meant typhoid fever with a localization of the typhoid bacilli in the lungs (see page 16), but it is also true that ordinary croupous pneumonia may occasionally appear as a complication of typhoid fever.

8. *Pneumonia with Delayed Resolution.*—While the resolution of pneumonia is complete, as a rule, in three days to a week after the occurrence of the crisis, there are cases in which this process demands a much longer time. Not infrequently, and particularly in severe cases of pneumonia, one sees after the crisis a surprisingly rapid disappearance of all the physical signs, while, on the other hand, recovery is sometimes remarkably slow in apparently mild cases; but this rule is, of course, not without exceptions. The course of the disease is often enough precisely the opposite. Just what are the conditions upon which the rapidity or the slowness of resolution depends we do not know. Sometimes unfavorable constitutional conditions, such as anæmia, debility, phthisical tendencies, and kyphoscoliosis, appear to delay resolution. Sometimes, on the other hand, no such explanation can be found. It seems to us that at certain times all the cases of pneumonia exhibit more of a tendency to delayed resolution than at others, so that it is not impossible that there are variations in the pathological process itself. In many cases of delayed resolution it is our opinion that we have to do with genuine secondary diseases, or a mixed infection of the lungs for the development of which the precedent croupous pneumonia had prepared a favorable soil.

With regard to the symptoms of delayed resolution, there are various forms. In the first place, we see cases where the crisis takes place in the usual way, and the temperature thereafter remains permanently normal. The patients perhaps feel quite well, and are troubled little by thoracic symptoms; nevertheless, the dullness upon percussion remains unchanged, or diminishes at best very gradually, and the bronchial breathing and moist râles can still be heard. All the signs diminish very slowly, sometimes occupying several weeks in their disappearance, and then complete recovery ensues. In a few cases it is striking that, after the crisis, bronchial respiration and dullness persist, while there are scarcely any râles and no expectoration. In these cases it seems as if the pneumonic exudation does not really liquefy at all, and is very slowly absorbed. In other cases there is no distinct crisis, but the fever continues, although lower than at first, and the physical signs persist to a greater or less extent. At the end of two or three weeks, or even still later, the fever slowly ceases, and thereupon normal resonance and vesicular breathing gradually return.

In still other cases the patients remain free from fever for the first few days after the crisis has taken place, although the pneumonia is not completely resolved. Then there appears again a rise of temperature which is mostly moderate, 100.5° to 103.5° F. (38° to 39.5° C.), while the dullness continues and there are an abundance of moist râles and catarrhal expectoration. After two or three weeks the fever gradually ceases and the abnormal pulmonary signs also slowly disappear. In such cases we may indeed suppose that some secondary infection, a sort of secondary catarrhal pneumonia, has developed upon the seat of the croupous pneumonia. The disease may take still another course somewhat different from those thus far described, and of this we have seen repeated instances much resembling one another. After the occurrence of the crisis the patient remains for about a week without fever. During this time the dullness and the not very loud bronchial breathing remain unchanged. Then appears a moderate intermitting fever, with elevations to about 102° or 103° F. (39.5° C.). This fever may last two to four weeks, or even longer. Over the affected portion of the lungs are heard either no moist râles or only a very few. Gradually there appears a moderate but distinct contraction of the side involved, then the resonance gradually grows clear, the respiratory sounds louder and vesicular, the fever ceases, and finally health is completely restored. In many other cases of delayed resolution, as we have already said, we see this same striking absence of moist râles and the development of a moderate degree of contraction. Under such circumstances it is often very difficult to exclude a secondary pleurisy, and we cannot make sure except by repeated exploratory puncture. Moreover, it is not exceptional to find delayed resolution and secondary pleurisy both present in the same patient.

9. *Termination of Pneumonia in Phthisis, Contraction of the Lungs, Pulmonary Gangrene, or Pulmonary Abscess.*—Four terminations of pneumonia are ordinarily mentioned as unusual and anomalous—the termination in "chronic pneumonia," in tuberculosis, in gangrene, and in abscess.

Concerning the termination in chronic pneumonia, we have already mentioned a process belonging here, the termination in contraction with ultimate recovery. In rare cases the contraction is permanent. The anatomical

process consists in the development of pulmonary cirrhosis, with the formation of a large amount of connective tissue. This proliferation, it should be said, takes place not only in the interstitial tissue, but also in the interior of the alveoli by extension from the alveolar walls. Few clinical observations of the further course of these cases, provided death does not shortly ensue, have as yet been published.

When croupous pneumonia is said to terminate in pulmonary tuberculosis, of course the statement can be understood to mean only that the symptoms of tuberculosis follow immediately upon an attack of pneumonia. When this is the case—it does not occur often—it is probable that the pneumonia attacked a person already suffering from tuberculosis, in whom, however, the symptoms did not become evident until after the pneumonia had run its course. It may also be that exceptionally the pneumonia furnishes a soil for a secondary infection with tubercle bacilli.

Pneumonia results in pulmonary gangrene in rare instances, when the patient is elderly, delicate, or diabetic. Here, too, in our opinion, a new infection, with a foul and putrid substance, must always take place, and this excites the gangrene. The previous pneumonia furnishes only the occasion for the development of gangrene, and perhaps prepares the soil for the agents of decomposition. The development of gangrene is appreciated clinically (see the appropriate chapter) by the change in the sputum and the persistent fever.

The transition from pneumonia to pulmonary abscess is very rare. We cannot decide whether a further specific cause is also needed for this, or whether the pneumonic process may exceptionally go on into the formation of abscess. We know that the pneumococci sometimes excite purulent inflammation in the pleura and in the meninges, and so it would not seem impossible that, under peculiar circumstances, they should also cause the development of an abscess in the lungs. The transition to an abscess may be recognized by the character of the sputum, which contains fragments of pulmonary tissue, such as elastic fibers, besides abundant pus. Moreover, we sometimes find, on microscopic examination of the sputum in abscess, scales of cholesterin (Fig. 49) and hematoidin crystals; the latter may be so abundant as to give the expectoration a brownish color. Sometimes we have seen a peculiar greenish color of the sputum. The signs of a pulmonary cavity are found if the abscess bursts.

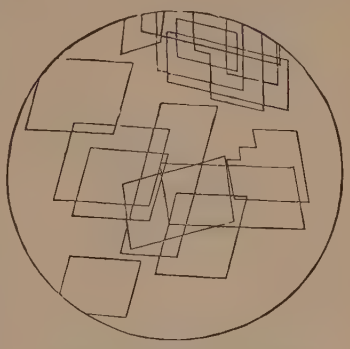

FIG. 49.—Cholesterin crystals.

Diagnosis.—No special remarks on diagnosis need to be added to the description we have given of all the important symptoms which may occur in croupous pneumonia. In the first place, we must consider the sudden onset with a chill and high fever, shortly followed by subjective thoracic symptoms, such as cough and pain in the side; also the characteristic sputum and the objective physical signs, the appearance in many cases of herpes on the face, and finally the general course of the disease, particularly the temperature

curve with its final sudden drop. We will discuss the differential diagnosis between pneumonia and pleurisy with effusion more fully in the description of the latter affection.

Prognosis.—Croupous pneumonia belongs in general to the benign infectious diseases. The great majority of cases, in previously strong and healthy individuals, run a favorable course, and end in complete recovery. On the other hand, pneumonia brings a number of perils with it, the knowledge of which should always make us cautious in giving a prognosis.

One grave danger lies in the extension of the process. If the advance of the pneumonia cannot be stopped, if the whole of one lung is involved, and, besides that, a great portion of the other lung, the diminution of the respiratory surfaces may of itself occasion a fatal termination.

A further danger lies in the onset of certain complications. An intense pleurisy, with effusion, especially if purulent, causes greater difficulty in respiration, and thus increases the danger. Still worse is a serofibrinous or purulent pericarditis, which, in not very rare cases, is revealed at the autopsy as the special cause of death. We must note, however, that recovery sometimes finally takes place in spite of an empyema or of a purulent pericarditis. The complication with a purulent meningitis, which is fortunately very rare, is probably invariably fatal.

The dangers of constitutional infection and constitutional intoxication are, on the whole, much smaller in pneumonia than in other infectious diseases; e. g., typhoid fever; but yet, this possibility deserves some consideration, particularly in certain forms of pneumonia already referred to and termed "typhoid" or "asthenic." Such particularly severe and malignant forms of pneumonia, with a high rate of mortality, sometimes appear as endemics and epidemics; but it should be added that these cases are also often marked by the extent of the local process and the development of the dangerous complications above enumerated.

The individuality of the patient plays the most important part in the prognosis of pneumonia. While a constitution that was previously healthy and uninjured usually survives the disease, one that was previously weak and diseased readily succumbs. In this fact lies the danger of pneumonia in old, weak, badly nourished persons, and in persons with a preëxisting emphysema, kyphoscoliosis, heart disease, etc. In this, too, lies the great danger of every pneumonia in drunkards. Since the nervous system is much affected by chronic alcoholism, we very often see outbreaks of delirium tremens in pneumonia. In like manner the other nerve centers are weakened and incapable of resistance, especially the regulatory centers for the heart and respiration. Hence we can understand why even moderate drinkers, though previously strong and well to all appearances, succumb to pneumonia from failure of the heart and impairment of respiration.

If we ask upon what symptoms our prognosis in any given case should depend, we must reply that no single factor can be given especial prominence. Chief stress must always be laid upon the state of the lungs and the respiration, but attention must also be given to the general condition, the heart's action, the height of the fever, etc. The worst dangers of pneumonia have just been mentioned.

Of the abnormal terminations of pneumonia, contraction gives the best

prognosis; but recovery, or at least a marked subsidence of all the symptoms, may sometimes take place after gangrene and abscess.

Treatment.—Many of the milder cases of typical pneumonia need no special active treatment when the disease takes a favorable course. Most cases get well under, or, we can almost say, in spite of any treatment. From the now obsolete method of treatment by large bleedings, and from the use of certain drugs (veratrin, tartar emetic), which are even now sometimes employed, we should expect harm rather than any benefit, yet under such treatment many patients have recovered.

We do not know of any certain means favorably to influence the pneumonic process. Whether we are destined to learn of some specific mode of treatment, perhaps by means of some serum, after the analogy of other infectious diseases, as diphtheria, we cannot say. A few beginnings have already been made in this direction, but without any practical results as yet. We are at present obliged, therefore, to fall back upon a purely symptomatic and constitutional treatment of pneumonia. [Of late, interest has turned to vaccination treatment, by injecting sterilized cultures of pneumonia bacilli, sometimes employing bacilli which have been grown from the patient's sputum (autogenous); but thus far the success has been partial or doubtful.]

The symptoms which are usually prominent in pneumonia, even in the milder cases, and of which the patient is especially desirous to be relieved, are the pain in the side, the troublesome cough, and the difficulty and distress in breathing. Since the respiratory symptoms, as we have seen, are partly due to the pain, as this improves the patient's breathing often undergoes a decided improvement. For the pain, we may first mention a number of external applications to the skin on the affected side. An ice bag sometimes gives marked relief. Many patients cannot bear this, but prefer warm poultices or cold wet compresses. The application of mustard plasters or dry cups to the skin may be of advantage. Subcutaneous injections of morphin, however, are the most effective remedy, and are often indispensable. There is no reason why we should not use this remedy, with care and in moderate doses, for the relief of pain; and, as the disease is of short duration, we need not particularly fear the morphin habit. Small doses of morphin, subcutaneously or by the mouth, may also be required to alleviate the cough.

Local bloodletting is a remedy the action of which cannot be explained physiologically, and yet experience has shown that it is of undoubted advantage. The relief which many patients feel after the application of eight or twelve leeches to the affected side is very striking; but we should prescribe them only when there are severe symptoms at the beginning of the disease, and in persons who were strong and healthy before the attack. Wet cups accomplish the same thing, but their effect is somewhat more powerful, and hence they should be reserved for strong and robust persons, such as laborers.

The tepid or cold bath serves as the most effective means of improving the respiration, of aiding expectoration, and of stimulating and refreshing the whole system. We hold it useless, if not injurious, to give a patient baths if the disease is progressing favorably, for almost every bath has some disagreeable feature. These disadvantages, however, are always less, in severe cases, than the benefit and improvement which baths give the patient, and which most patients recognize with gratitude. The main point is that the patient

should make no physical exertion while in the bath, that he should be lifted into it, held and supported while in it, and lifted into bed again after it. Since the baths are given primarily not on account of the fever, but to improve the respiration, and because of their favorable influence on the nervous system, their temperature need not be especially low. We give them from 77° to 86° F. (25° to 30° C.); somewhat warmer with sensitive and weak people, and cooler, down to 73° F. (20° C.), with strong persons, or with very high fever or severe nervous symptoms. We need not employ more than two or three baths a day, and at night we employ them only when there are threatening symptoms. The favorable action of the baths is seen especially in the great relief and refreshment that the patient feels. The respiration is quieter and slower, but deeper. The patient often falls into a quiet sleep after the bath. Of late years we have often replaced the baths by a wet pack, even in adults. This has been almost always very well received, and indeed seemed to make the patients quieter, with easier respiration and less discomfort. We would particularly and strongly recommend the pack for private patients, in whose case the employment of baths is associated with many difficulties.

Among internal remedies for pneumonia, antipyretics are frequently employed. We do not believe they are capable of exerting any decided influence upon the general course of the disease, although we admit that antipyrin and, under some circumstances, also phenacetin and antifebrin have a good effect, since they not only modify the fever, but improve the nervous symptoms and the general condition. Antipyrin is prescribed in doses of 15 to 30 gr. (gm. 1 to 2), particularly toward night. [It is seldom that antipyretics are justified in pneumonia.]

To make the cough somewhat more effective, expectorants are prescribed. We ourselves most frequently employ infusion of ipecac, apomorphin, infusion of senega, liquor ammonii anisatus, and benzoin. The last two remedies are especially favorable if the heart is feeble. It also seems to us of some importance, with regard to expectoration, to prescribe an abundance of liquid in the form of water, tea, lemonade, and similar drinks.

The behavior of the heart should always be watched with vigilance, especially in elderly and delicate persons, and in the obese and alcoholic. If the pulse becomes very frequent, an ice bag is placed upon the heart. If the pulse rate is remarkably rapid from the start, we may order digitalis at once, either in infusion, or, still better, in powders containing 1.5 to 3 gr. (gm. 0.1 to 0.2), repeated several times a day; and as digitalis acts slowly, requiring twelve to twenty-four hours to make its influence felt, if the weakness of the heart is dangerous, stimulants which act more promptly must be employed. As such, caffein and tincture of strophanthus are to be recommended, and, above all, subcutaneous injections of oleum camphoratum (1 to 4), of which three or four syringefuls (♏ xv), and more, may be employed (ʒss. to ʒj). Again, ether injected subcutaneously is a powerful cardiac stimulant, but it should be employed cautiously because of its marked local effect (tissue necrosis and paralysis of the muscles), and the place of injection should be the skin of the abdomen or the thigh.

We have yet to make some remarks upon the very extensive use of large amounts of alcohol in pneumonia. A free use of alcohol is generally considered necessary in drunkards, especially when delirium tremens is beginning

or is already pronounced. Since the withdrawal of any poison that is taken habitually, like nicotin or morphin, may excite the severest symptoms, the sudden withdrawal of alcohol from drunkards may have the worst results, while, if we give an abundant supply of the stimulant to which the nervous system is accustomed, we sometimes succeed in avoiding the onset of severe nervous symptoms, such as collapse and failure of the heart and respiration. It is questionable if this view is correct. All in all, I have not gotten the impression, from my personal experience, that the withdrawal of alcohol leads to any serious manifestations. I shall not, however, question the propriety of the exhibition of alcohol in the pneumonia of alcoholics, but I, myself, never insist upon the same. Likewise wine should be given to persons who are accustomed to it and themselves desire it. It is quite a different matter with patients who before their illness have not been accustomed to take alcohol at all, or who took it only in small amounts. It may be true that in these cases moderate amounts of wine may have a stimulating and exciting action, although we never could satisfy ourselves of the often praised influence of alcohol upon the action of the heart. We hold it, however, unjustifiable to force large amounts of alcohol indiscriminately upon every patient with pneumonia, perhaps in spite of great resistance on his part. Why should we expect sick persons to be benefited by doses of alcohol which have only bad results on healthy men unaccustomed to them? The assertion that fever patients "bear" alcohol better than healthy persons lacks proof. It should be admitted that perhaps alcohol is more rapidly consumed in fever than in health, but it should also be considered that the toxic effects of alcohol are less easily noticeable in comatose patients than in persons possessing normal consciousness.

[Probably a moderate amount of alcohol may contribute to the support of a patient who is beginning to fail in pneumonia, but it is not wise to give very large doses, even if the patient is very feeble. Better are strychnin given subcutaneously in full doses, but stopping short of physiological effect, and caffein also subcutaneously. If the pulse tension is low and the abdomen distended, a flannel-covered ice bag placed on the abdomen may act favorably by stimulating the splanchnic nerve. Oxygen inhalations do not seem to be of very great value, much less so than in broncho-pneumonia. Emphasis must be placed on fresh-air treatment. Apparently a low temperature makes the air better. The patient should be warmly but lightly covered. He may be in a room with the windows all open, or, still better, actually in the open air. The benefit is fully as great with children as adults. Speaking of the Baby's Hospital, in New York, LaFetra says: "1. In the acute congestive stages of inflammations of the upper respiratory tract (rhinitis, laryngitis, bronchitis, especially of the small tubes, with or without bronchial pneumonia) warm, moist air is of greater help than cold, dry air. 2. In lobar pneumonia, with high temperature and little or no bronchitis, cold, dry air is of great advantage. The same is true of pulmonary tuberculosis and of emphysema. 3. After the acute stage has passed, and when there is no inflammatory spasm of the larynx or bronchi, the cold-air treatment is of advantage in cases of rhinitis, laryngitis, bronchitis, and bronchial pneumonia."]

We scarcely need to lay special emphasis on the fact that the physician should endeavor, so far as possible, to maintain the bodily strength by means of appropriate and sufficient nourishment. Soups, bouillon with toast or rusk,

milk, and eggs are the most suitable articles of diet, and it is sometimes proper to give small amounts of finely minced meat. Care should be taken that the patient has an abundance of refreshing beverages. Wine and also moderate amounts of good beer may be allowed without hesitation.

The treatment of complications follows the ordinary rules which have been given under the individual affections. We must also mention that, in delirium tremens, tepid baths with cold douches sometimes have a very good effect. Besides this, we may try subcutaneous injections of strychnin, 7 to 15 minims of a one-per-cent solution once or twice a day. We cannot wholly dispense with narcotics, such as morphin and chloral, but we must warn against the imprudent use of large doses of chloral, above 35 gr. (gm. 2.5). Paraldehyd, in doses of 45 to 75 minims (gm. 3.0 to 5.0), is to be recommended.

CHAPTER VI

TUBERCULOSIS OF THE LUNGS

(Pulmonary Phthisis. Pulmonary Consumption)

GENERAL PATHOLOGY AND ÆTIOLOGY OF TUBERCULOSIS

Ever since Bayle, in 1810, demonstrated the extensive distribution of peculiar nodules in the various organs, and their relation to pulmonary consumption, few questions have so taxed clinical observers and pathologists as those relating to the cause and nature of tuberculosis. Harmony could not be reached, however, so long as the criterion for the decision of the questions was sought in the presence of definite anatomical changes, which were regarded as specific of tuberculosis. Laennec considered the peculiar change in the tuberculous products, which later was named caseation by Virchow, to be characteristic, and called everything tuberculous in which it was found. He distinguished the isolated tubercle from diffuse, tuberculous, cheesy infiltration. Thus, Laennec recognized that many processes were allied whose affinity was often disputed afterwards, and which has only recently been established, such as the affinity between "scrofulous" enlargement of the glands and tuberculosis. Another opinion became quite prevalent, after Virchow discovered that precisely the same anatomical process as tuberculous caseation was also found in inflammatory products, which were certainly not tuberculous, and in cancerous ulcerations. Hence, Virchow made a sharp distinction between tubercle and those new growths and inflammatory processes which had become cheesy. The anatomical criterion of tuberculosis was, in his view, the presence of the miliary tubercle, a gray nodule, the size of a millet seed at the largest, made up of cells like lymph corpuscles. The study of the finer structure of the miliary tubercle was now pushed most eagerly by Wagner, Schüppel, Langhans, and others, but they were unable to reach perfect harmony regarding its origin and significance.

As long ago as 1865, however, a discovery was made which pointed unequivocally to the only way which could lead to a correct knowledge of tuberculosis. It was the fact, discovered by Villemin, that tuberculosis can be pro-

duced artificially by inoculating healthy animals with small amounts of tuberculous and cheesy substances. Although doubted and misinterpreted at first in various quarters, the fact that tuberculosis can be transmitted, and consequently the fact of its infectious character, must now be regarded as proved beyond a doubt. In the general change which our opinions upon the nature of infectious diseases has undergone, especially in the last few years, the existence of a specific, organized cause of tuberculosis, too, had to be assumed. Klebs, and later Cohnheim, had already without reserve defined tuberculosis as a specific, infectious disease, and, sooner than we dared to hope, R. Koch discovered the special carriers of the disease in the shape of the tubercle bacilli, in the year 1881. The definition of tuberculosis no longer rests upon any external anatomical character. Every disease is tuberculous which is excited by the pathogenic action of a specific kind of bacteria, the tubercle bacilli discovered by Koch.

The pathogenic bacteria of tuberculosis belong to the group of bacilli. The tubercle bacilli are rod-like, of small diameter, slightly rounded at their extremities, and either straight or somewhat bent. Their length is perhaps a fourth or a half that of the diameter of a red blood corpuscle. In the interior of these rods it is not infrequently possible to distinguish very minute colorless spots which are probably to be regarded as endogenous spores. The tubercle bacilli have no independent motion whatever. Their reaction to certain coloring matters is very characteristic, and of the highest importance with regard to their recognition (*vide infra*).

We know with absolute certainty that the tubercle bacilli are always present in all the different forms of pulmonary tuberculosis, both in the lung itself and in the expectoration (*vide infra*), and also in tuberculous diseases of other organs, the brain, the intestines, the spleen, the liver, and the kidneys, and also in "scrofulous lymph-glands," in "fungous" diseases of the bones and joints, and in the so-called lupus, which is nothing but a local tuberculosis of the skin. Precisely the same bacilli are also found in the "spontaneous" tuberculosis of animals, such as monkeys, puppies, and guinea pigs, and in every tuberculosis that is artificially produced in animals by inoculation. Finally, by the discovery of tubercle bacilli in the "pearly distemper" of cattle, the close relationship of this disease with tuberculosis has been established.

Koch, by his successful "pure cultures" and inoculations with the cultivated bacilli, has established the fact that these tubercle bacilli are to be regarded as the cause of tuberculosis. Bacilli coming from any fresh product of tuberculous disease may be grown in pure culture at a constant temperature of 98° to 100° F. (37° to 38° C.) upon blood serum which has been stiffened by heating, and upon several other artificially prepared soils. In this cultivation they show certain characteristic properties in their growth, which cannot be fully described here, and they multiply to an unlimited extent. By the subcutaneous injection of the smallest amounts of sputum containing tubercle bacilli into guinea pigs, the local lymph-nodes first become swollen, and after about thirty days tuberculosis of the spleen is to be found, and after some forty days tuberculosis of the liver. Death occurs seven to eight weeks after the injection, during which period the animal becomes markedly emaciated. If the animals inhale the infectious material, numerous tuberculous foci are to be found in their lungs about three weeks later. Tuberculosis may likewise

be developed in animals fed with tubercle bacilli, but for this purpose, however, exceedingly large quantities of the infectious material are required. According to Flügge, 400,000,000 bacilli, in a single feeding, are an effectual dose for a guinea pig. The most instructive inoculations are those into the anterior chamber of the eye in puppies or guinea pigs, which were first tried by Cohnheim and Salomonsen. After an incubation of two or three weeks we see here very plainly an eruption of the first nodules of tubercle in the iris, and the tuberculosis spreads to the other organs of the body later. By all of these experiments it was demonstrated that tuberculosis is always, at the start, a purely local disease, and that its further extension depends upon the spread of its germs.

Ætiology of Tuberculosis in Man

The distribution of tubercle bacilli must be remarkably extensive, for tuberculous diseases occur in almost every country on earth. The predisposition of mankind to the disease is also very great, and thus we understand the frightful fact, which statistics show, that about one eighth of all deaths are from tuberculosis! The number of individuals who are at all infected is very much larger still. According to some statistics, tuberculosis is present in from eighty to ninety per cent of all post-mortem cases. The greatest number of these cases, to be sure, show healed lesions. It has neither been proved, nor is it probable, that tubercle bacilli multiply outside of the human body, like the bacilli of splenic fever, since they can develop only in a constant and uniformly warm temperature between 85° and 104° F. (30° to 40° C.). We must therefore regard them as true parasites, which can live—that is, which can propagate and multiply—only in the bodies of animals, but they (or their spores) seem to preserve their virulence and their ability to multiply outside of the body for a long time. Phthisical sputum may be used for inoculation with success, even if it has been dried for several weeks. The tubercle bacilli also resist most chemical reagents, such as nitric acid, very decidedly.

If the body becomes infected, then, with tubercle bacilli, it is always probable that they have come from some other individual—man or beast—with tuberculous disease. We need not mention how numerous the opportunities for infection may be, considering the present general distribution of tuberculosis. The chief stress in this respect is very evidently to be laid upon the expectoration of phthisical patients, which contains the bacilli. The extensive investigations of Cornet have well shown how frequently it is possible to obtain tuberculous material, which is capable of causing infection, from the dust in the neighborhood of phthisical patients who carelessly deposit their expectoration upon the floor and in handkerchiefs and elsewhere. On the other hand, Flügge's studies have shown that dried sputum does not readily become pulverized and whirled about in dust. Tubercle bacilli were only exceptionally found in the dust gathered at a height of about that of an adult's head from the floor in waiting rooms, factories, street cars, etc. For these reasons, Flügge considers another mode of infection to be of greater significance. He has demonstrated that the consumptive with a cough can defile the surrounding air with numerous minute drops of sputum containing tubercle bacilli. These hover for a time in the air, and may be inhaled by other persons. This

mode of infection is frequently important in the direct transmission of tuberculosis, and such direct transmission is shown, above all, by the cases of tuberculosis in married couples, in nurses, and in patients who for a long time occupy a ward in which there are many consumptives. Still, the experience of physicians proves that such direct infection with tuberculosis is of very infrequent occurrence (*vide infra*). There are very many who fall sick with tuberculosis who have never been in close contact with other consumptives. In such cases it must be that the tubercle bacilli enter the body indirectly. This may naturally occur in the most diverse ways. The tubercle bacilli may be scattered by the diseased individual by means of the sputum itself, as well as by the expulsion of drops of mucus containing bacilli during coughing.

The material which contains the bacilli or spores is taken into the body, in the majority of cases, along with the inspired air. This is probable, because, in most cases, tuberculosis has its starting-point in the air-passages, the lungs, or the larynx. Inoculation experiments show, as we have indicated, that the first extension of the process depends upon the point of inoculation. Hence it seems very probable that, in tuberculosis in man, the infectious matter is taken directly into the air-passages by the breath. In this way it sometimes, though rarely, attacks the upper air-passages, as in primary tuberculosis of the nose, the pharynx, and the larynx, but more frequently it affects the deeper portions of the respiratory apparatus, as in primary tuberculosis of the lungs and bronchi.

It cannot be denied, however, that even in the presence of an apparently primary pulmonary tuberculosis, the possibility of an indirect infection of the lungs cannot be absolutely excluded. Certain experiments seem to show that tubercle bacilli that have perhaps first passed by way of the tonsils or through small wounds of the skin into the neighboring lymph-glands, finally reach the lungs via the blood or lymph-channels, where they at last find conditions favorable to their settlement and to the development of their harmful effects. At the present time it can by no means be definitely stated to what extent the direct, and to what extent the indirect, mode of infection plays a part in primary pulmonary tuberculosis. The fact that pulmonary tuberculosis is so often apparently first situated at the apex, and that, on the other hand, the lower lobes are most often involved in all the other inhalation diseases of the lungs, may be advanced as an argument in favor of the indirect infection of the lungs. The important X-ray examinations (*vide infra*) that have been made in incipient tuberculosis (H. Rieder) have, however, led to the interesting result that in many cases, at least, the apices are by no means the first seat of the disease, but are only involved during the second stages of tuberculosis. The bronchial lymph-glands are primarily affected, an observation clinically not demonstrable without X-ray examination. In the X-ray picture strands (probably, for the most part, tuberculous lymphangitis) may be seen extending from the lymph-nodes to one or both of the upper pulmonary lobes, whence the disease establishes itself and spreads further. In most cases the bronchial glands certainly become diseased through inhalation of the tuberculous virus. Here the latter first passes through the lungs, leaving them unaffected, and only being taken up by the bronchial glands, where it remains. (Compare with the chapter on Pneumonoconiosis.)

In addition to infection through the atmosphere, the possibility of infection

of the intestinal canal from the swallowing of infectious material must be considered. The transmission of tuberculosis from domestic animals to man plays a part in this connection which perhaps is not unimportant. Since the pearly distemper of cattle is identical, or at least closely related to tuberculosis in man, the use of the flesh of such animals as food furnishes a possible means of infection. It is a still more important circumstance that, when pearly nodules are present on the udder, the milk of the diseased animal may be polluted by tubercle bacilli, and that the use of such uncooked milk or butter made from it as food certainly involves the danger of the transmission of tuberculosis. Indeed, Behring has expressed the opinion that the greater proportion of cases of pulmonary tuberculosis are referable to an infection of the intestinal canal dating from earliest childhood. This assertion is not in accordance with general experience, and has met with but little approbation in medical circles. It is all the more improbable because Koch, Kossel, and others have demonstrated that the bacilli of human and of bovine tuberculosis are not identical. Two different types of the tubercle bacillus, the *typus bovinus* and *typus humanus,* can be readily distinguished. Human tubercle bacilli usually produce only a local tuberculosis in cattle, whereas a general tuberculosis may readily be given to them by inoculation with the bacillus of bovine tuberculosis. On the other hand, the bovine bacilli do not seem to have any great virulence for human beings. That some virulence exists, however, is shown by some cases of intestinal and peritoneal tuberculosis in children, in which just the bovine type of bacilli can be demonstrated. The danger of milk infection, especially in children, cannot therefore be overlooked, but its importance should not be overestimated. In most cases, tuberculosis in children certainly does not begin in the intestine, but in the lymph-nodes, and, above all, in the bronchial nodes surrounding the roots of the lungs.

In some cases the tuberculous infection may probably arise from little fissures and excoriations of the skin. In this way either we get a local tuberculosis of the skin, such as lupus, or the bacilli are carried by the lymphatics to the neighboring glands of the neck or axilla, establish themselves there, and set up a tuberculous disease in them. It has been pointed out above that tubercle bacilli may also pass from the lymph-nodes into the blood and thus reach distant organs. Because of its importance, it should again be stated that even when the tubercle bacilli are taken into the body through the lungs or the intestine, the infectious material often passes the primary seat of infection without fastening itself there, and does not take root until it reaches the nearest lymph-glands; or perhaps, often, still more distant regions, such as the kidneys or the bones (?). Thus arises the so-called primary tuberculosis of the bronchial or mesenteric glands, leading sometimes, as we shall see later, to various severe tuberculous diseases in other parts, such as the pleura or peritoneum.

It remains to mention the possibility of a primary genito-urinary tuberculosis. Primary infection of the uterus and ovaries might be referable to direct infection from without; but it is questionable whether genito-urinary tuberculosis of the male, affecting the kidneys, testicles, and prostate, arises in this manner. In such cases we are probably generally dealing with infection due to the excretion by the kidneys of tubercle bacilli which have got into the body in some way, and have reached the general circulation.

Considering the wide distribution of the tubercle bacilli, and the many chances for infection, it seems wonderful that in spite of it so many human beings escape the disease. One factor, which has been already mentioned by Koch, must be borne in mind, however, and that is the extremely slow growth of the tubercle bacilli. This is the reason why the bacilli do not always remain in the body, but in many cases are eliminated again before they have gained a definite foothold.

Individual predisposition, however, is another factor which is probably still more important—a factor which we cannot well explain, but which we cannot get on without, at the present time, in the pathology of many infectious diseases. In our conception of most of the other infectious diseases as well as of tuberculosis, we must assume provisionally an unequal predisposition to disease in different individuals. Of all individuals who are exposed to the invasion of tubercle bacilli, only those become diseased in whom the poison can establish itself in the tissue cells where it exerts its deleterious influences, and whence it further increases and spreads.

The basis for this "predisposition to tuberculosis" is not well understood. According to our present views on the general principles of immunity, however, it is by no means impossible that the variation in the predisposition to tuberculosis is at least in part dependent on the varying individual, chemical state of the blood serum and the tissue juices. In this connection, the recent investigations of R. Koch, Behring, and others have taught that there are decided differences in the agglutinating effect of the serum of tuberculous and of nontuberculous individuals on cultures of tubercle bacilli. Yet it is very striking that the existence of the tendency to tuberculosis is often expressed in the general constitutional debility of the individual, and—still more remarkable—in certain peculiarities of his bodily frame, e. g., the shape of his thorax. This phthisical habitus (*vide infra*) is especially common in persons who come from families with a tendency to tuberculosis, and it is a peculiar yet quite inexplicable expression of the predisposition to tuberculosis, due to family and hereditary influence (*vide infra*). It should be said that the hereditary tendency to tuberculosis is not invariably associated with any striking and self-evident weakness of the constitution. Persons who spring from tuberculous families may have a vigorous frame and yet fall victims to the disease; and likewise, also, when there is no hereditary tendency, the most powerful build does not by any means render one invulnerable to the disease.

We now believe that many evil influences which were once thought to be causes of tuberculosis act only in increasing the predisposition to the disease. Insufficient food, bad air, severe illness, childbirth, want, and care—these alone, of course, can never produce tuberculosis; but innumerable observations show that the body which has become weakened affords less resistance to the injurious influence of the tuberculous poison than does the strong and healthy body. Thus it seems to us, from our own observations, to be in the highest degree probable that chronic alcoholism increases the liability of the individual to tuberculosis. It is surprising how often drunkards, possessed of a naturally most vigorous constitution, fall victims to tuberculosis.

People used often to speak of the transition of other affections of the lungs into pulmonary consumption—that is, into tuberculosis. It was imagined that an old bronchial catarrh, croupous inflammation of the lungs, or the

catarrhal pneumonia accompanying measles, or whooping cough, could readily become "tuberculous." Of course the proper interpretation of such a connection is that the precedent disease prepares a favorable soil for infection with the tuberculous virus, and that consequently the tubercle bacilli fasten more readily upon the mucous membrane, which has been previously affected, than under normal conditions. Moreover, many of the infections which we formerly supposed to be apt to change into tuberculosis are themselves tuberculous. This is true in most of the so-called scrofulous diseases of the lymph-glands, bones, etc., and also, as we shall see later, in the overwhelming majority of cases of apparently primary pleurisy.

No single factor, however, of those which favor the predisposition to tuberculosis, plays so manifest and so visible a part as does the hereditary tendency above mentioned—that is, the inborn predisposition of the individual. The fact of the heredity of phthisis meets us with such uncommon frequency that it must have forced itself upon the notice of the older physicians. In the great majority of all cases of phthisis we can make out, by close questioning, that in the family, either among the older members or among the brothers and sisters, one or more cases of tuberculous disease have already occurred. The closer we investigate, and the more we search for some one of the different forms in which tuberculosis can show itself, like pleurisy, or affections of the bones and joints, the more frequently we can make out this hereditary predisposition. Some persons are indeed of the opinion that the hereditary transmission of the disease is often merely apparent, and not real, from the fact that the close relations between the children and their diseased parents, or brothers and sisters, greatly increases the danger of infection of the ordinary kind. Certainly this consideration should not be forgotten with regard to the occasional appearance of tuberculosis in families, but yet it would be impossible to explain in this way alone the extremely frequent development of tuberculosis in special families.

There can be, therefore, scarcely any doubt as to the inheritance of tuberculosis, but the explanation of this fact is far from clear. The hereditary character of tuberculosis may very well harmonize with its infectious character. We might assume in this case a perfect analogy with syphilis—namely, a transition of the infectious material itself from the parent to the child before birth. There is only one striking difference between syphilis and tuberculosis —that the children of syphilitic parents very often come into the world with definite signs of infection, while congenital tuberculosis in this sense is an extremely rare occurrence. We must accordingly compare tuberculosis to that form of hereditary syphilis (*lues hereditaria tarda*) in which the first symptoms of the affection come on at a late period. Since certain considerations, however, oppose such a theory, we are of late far more disposed to assume that, as a rule, tuberculosis in itself is not inherited, but only the predisposition to tuberculous disease. This opinion agrees with the facts that members of a family in which tuberculosis prevails very often show the so-called tuberculous habit even without any real tuberculous disease; and that they often have "weak lungs"—that is, that they easily get out of breath, and manifest a distinct tendency to catarrh of the respiratory organs. Another fact, which to a certain extent may be regarded as an argument against the assumption of a direct hereditary transmission of the disease, is that, in

cases of apparently hereditary tuberculosis, as a rule those organs first show evidence of disease which are most exposed to an infection from the outer world—namely, the lungs and larynx. This is also an argument against the acceptance of an exclusively hereditary predisposition to tuberculosis.

The age of the patient has an important influence upon the predisposition to tuberculous disease. Pulmonary tuberculosis occurs with special frequency in youth, between fifteen and thirty. It is not rare in children. After forty it is much rarer in its pronounced and rapid forms, but it is seen even in the most advanced age. Slight tuberculous changes are very frequently found at autopsy in the lungs of old persons. These changes have, as a rule, no clinical significance. They are in all probability due to the inhalation of tuberculous germs with scarcely any tendency to further extension because of the lack of individual predisposition to the disease. It has not yet been shown that sex has a special influence upon liability to the disease.

PATHOLOGICAL ANATOMY OF TUBERCULOSIS, ESPECIALLY OF PULMONARY TUBERCULOSIS

If now we inquire wherein consists the injury which the tubercle bacilli inflict upon the body, the first point to emphasize is that the action of the tubercle bacilli is at first invariably purely local. Tuberculosis does not belong to the "general infectious diseases," in which the infection of the whole organism, the general infection of the body, predominates over the local disturbances. The essence of tuberculosis, at least in the great majority of cases, is the local disease. The tubercle bacilli give rise to definite anatomical changes in the organs where they settle, and the consequent disturbance of function in the organ has an effect on the rest of the body.

The danger of tuberculous diseases, however, consists in the fact that the local affection often attacks the most important organs, as the lungs and the brain, and sets up such extensive anatomical changes in them that because of these changes alone it becomes impossible for life to continue longer. Besides this, the infection in many cases does not always confine itself to one organ, but the infectious material extends over the body by ways and means which we shall learn in part later on, and attacks one organ after another, or even many at once. Finally, it is to be said that the peculiar character of the changes caused by tuberculosis explains why these are often the cause of manifold secondary processes, particularly secondary infections. Thus arise fever, suppuration, secondary inflammation, and other important phenomena, which will be minutely considered later.

The entire local action of the tubercle bacilli—that is, the pathological anatomy of tuberculosis—is almost wholly independent of the organ attacked. Tuberculosis belongs to the group of so-called "infectious tumors"—that is, the local action of the tubercle bacilli consists chiefly in the production of a proliferation and accumulation of cells at their place of settlement, which, as a whole, is termed a tubercular new growth. Without going into histological details we may say, briefly, that the whole process consists in a primary injury of the tissue, due to the invasion of the tubercle bacilli, followed by a hyperplasia of the cells peculiar to the tissue itself, and involving not only the connective-tissue cells and the endothelial cells of the lymph and blood

vessels, but also, possibly, the epithelial cells. The so-called epithelioid cells, which are large cells, rich in protoplasm, and the multinucleated giant cells, most often found in the center of tuberculous nodules, are derived from the hyperplasia of the connective-tissue cells. The giant and epithelioid cells are surrounded by numerous round cells. These, at first, are ordinary leucocytes, for the most part of the mononuclear lymphocyte type, and then usually later on migrating polymorphonuclear leucocytes appear. The round cells referred to may, in some cases, finally include nearly the entire tubercle. A network, or reticulum, is found between the individual new-formed cells and the wandering cells. This probably represents the remains of the original interstitial tissue, crowded apart by the increased number of cells. There is no new formation of vessels. The tubercle contains no blood vessels. The tubercle bacilli lie especially inside the giant cells, but also in their vicinity.

If these changes have progressed far enough, they become visible to the naked eye as a small, circumscribed grayish point, the so-called miliary tubercle. From these minute nodules the disease itself obtained its name of tuberculosis. By approximation and coalescence of neighboring tubercles—for these keep developing because of the spread of the local infection—the tubercular formation continually extends itself, and thus are gradually formed the large tubercular nodules, and finally the diffuse tubercular new growth or the diffuse tubercular infiltration.

The tubercular new growth, as such, differs but little histologically from other infectious tumors, such as those seen in syphilis and leprosy. Tuberculosis, however, has a characteristic difference in its final stages of cheesy degeneration and eventual disintegration of the new-formed tissue, processes which are connected in part with the absence of blood vessels and the consequent deficiency of nutrition of the new formation, in part with the toxins that are elaborated by the tubercle bacilli. Both the tubercular infiltration and also the portions of the original tissues inclosed by it perish, lose their nuclei, and finally become disintegrated. The manner in which they are destroyed—namely, "fatty degeneration"—belongs in the group of the so-called coagulation necroses (Weigert). This process is recognizable to the naked eye, because the tubercular infiltration when it becomes thus degenerated takes on a pronounced yellowish color. Wherever the necrotic portions of tissue are superficially situated they are cast off, giving rise to tubercular ulcers.

Alongside the tubercular new formation there are found in the organs affected with tuberculosis various inflammatory processes, either simple or suppurative or hemorrhagic. We may therefore surmise that the tubercle bacilli (or the chemical toxins formed by them), besides their characteristic effects, act simultaneously in another rôle as excitants of inflammation; but it is very probable, and especially so in pulmonary tuberculosis, that many of the inflammatory changes which arise are not peculiar to the tuberculosis as such, but are to be regarded as secondary processes (*vide infra*) for the development of which the preceding tubercular new growth, as it disintegrates, furnishes a suitable soil.

As regards the special anatomical processes and appearances in pulmonary tuberculosis, the tuberculous change usually begins in the walls of the smallest bronchi, or not rarely in the alveoli themselves. At any rate, the disease does

not begin in many different parts of the lung at once, but probably in one or two circumscribed spots only, and in a great majority of cases in one apex. The tubercular infiltration begins in the walls of the bronchi, and thence gradually extends toward the periphery. The original tuberculous bronchitis becomes a tuberculous peribronchitis. The infectious material extends from its original focus by means of the lymph and blood channels out into the surrounding tissue; and also as soon as superficial ulceration takes place, the infectious matter is liable to be carried by the inspired air into other bronchi. Thus the diseased spot, small at first, keeps gradually extending. Tuberculous peribronchitis is usually easily recognized with the naked eye. We notice the little lumen of the bronchus in the middle of the "cheesy" nodule, which at first is gray and later yellowish. Many of the adjacent nodules run together in part, and even entirely. The lumen of the bronchus is either wholly plugged by the infiltration, or the destruction of the necrotic cells begins in the midst of the peribronchitis. In the latter case the lumen is enlarged to a little irregular hole—the first beginning of the formation of a cavity.

The alveolar tissue of the lung cannot long remain unaffected, with such a disease of the smaller bronchi. Lobular atelectasis, the necessary result of every permanent bronchial obstruction, must arise, but this soon passes (by penetration or aspiration of the germs) into a lobular pneumonia, which from its specific nature later becomes caseous. We cannot go into the histological details here. The alveoli are filled with pus corpuscles and large epithelioid cells, which are considered by many authors to be the offspring of the alveolar epithelium. The alveolar walls are also infiltrated. This finally results in the destruction of the cheesy and necrotic tissue, and consequently in the formation of cavities. At other times the neighboring nodules run together, and the tubercular infiltration thus extends, giving rise to a diffuse caseous pneumonia. These processes may all be readily recognized by the naked eye. The earlier stages of atelectasis and infiltration correspond to the jelly-like, gray coloring seen in the so-called gelatinous infiltration of Laennec, and the transition to caseation is recognized by the eye from the yellowish color.

Although all the processes thus far described are destructive in their nature, changes are also found in the lungs in tuberculosis which seem to have a tendency toward circumscribing the disease and toward healing. Prominent among these are the chronic interstitial processes. We meet with the formation of new connective tissue, partly about the tubercular infiltration, but especially where there is already destruction of tissue, and this may lead to contraction and the formation of a firm cicatrix. The encapsulated cheesy masses may then be in part reabsorbed; in part they undergo calcification. The possibility of such a halt in the tubercular process depends, however, upon certain conditions. The tubercular new growth and its destruction must not advance too rapidly, and the new-formed tissue must not itself be destroyed before it becomes cicatrized. We see the cicatricial formation, therefore, more especially in chronic cases; we find it in places which have been affected the longest, and where the tubercular process, perhaps, has finally come to a standstill of its own accord. Macroscopically, this cicatricial connective tissue is composed of a thick, firm substance, usually pigmented—the so-called pigment induration. If the cicatricial formation follows a previous

extensive destruction of the pulmonary tissue, the affected portion of the lung may thus be diminished to less than half its original bulk. Cavities and firm cicatricial tissue form the anatomical basis of such an extensive "pulmonary contraction." Either the cavities are formed in the usual way from the destruction of lung tissue, or they may be simple bronchial dilatations due to the traction of the contracted tissue—bronchiectatic cavities.

The contractile changes in pulmonary tuberculosis teach us that the tubercular process is in itself capable of healing. The incurability of most cases of phthisis is due to the fact that the infectious material from every existing tubercular nodule is carried into other bronchi, and there sets up a new tuberculosis. Thus the disease is constantly extended. The original tuberculosis, which was localized in one apex only, gradually spreads to the lower portion of the lung. The infectious material is carried by coughing into the trachea, and from this point may be carried by inspiration into the other lung. This becomes diseased, and finally there is such an extensive destruction of the lungs as to make the further continuance of life impossible.

Besides the specific tuberculous lesions in phthisical lungs, there are very often found simple inflammatory processes, such as bronchitis and lobular catarrhal pneumonia; sometimes, also, although seldom of great extent, croupous pneumonia; and finally, in exceptional instances, limited foci of gangrene. These secondary diseases not specifically tuberculous, and yet almost always associated with tuberculosis, are of the greatest clinical importance. They probably are due in most instances to the influence of secondary pathogenic germs (particularly streptococci, less often diplococci, etc.), for which the tuberculosis has merely prepared a favorable soil. Many clinical phenomena (particularly most of the febrile exacerbations of the disease) depend upon these secondary inflammatory processes which, in their turn, promote the further extension of the tuberculosis. Thus, frequently, inflammatory lesions which are due to secondary infection finally are transformed by the invasion of tubercle bacilli into foci of tuberculosis.

If we consider the list of anatomical processes which are found in tuberculosis of the lungs, and which may be combined in the most manifold ways, we can understand the great diversity in the anatomical picture in different cases. Primary tuberculosis of the bronchial wall and tuberculous peribronchitis, diffuse cheesy pneumonia, and destruction of the tubercular new growths, with the formation of cavities, on the one hand; contracting interstitial pneumonia, cicatricial formation, and pigment induration on the other —these are the comparatively simple anatomical lesions from which the whole process of pulmonary tuberculosis in its most varied forms is composed. Besides this, we often find here and there one or more miliary tubercles scattered through the lungs, which are probably due very largely to an extension of the infectious material by means of the blood or lymph current; and finally the already mentioned processes of secondary inflammation, such as bronchitis and pneumonia.

The secondary tuberculous diseases of the pleura, the bronchial glands, and other organs, will receive a special description.

CLINICAL HISTORY OF TUBERCULOSIS IN GENERAL AND OF PULMONARY TUBERCULOSIS IN PARTICULAR

In judging of the various appearances in the clinical picture of tuberculosis we must especially consider the following points: The place of the first infection is of chief importance—that is, the place where a local affection, set up by the tuberculous poison, first arises. One can readily understand why all those organs which are directly exposed to infection from without are most frequently affected with primary tuberculosis. Very often, as we have said, the lungs are the organs first attacked. Less often the upper respiratory passages—viz., the larynx and nose. In other cases the tubercle bacilli fasten first upon the digestive tract (the intestines, less often the pharynx and the tongue). In many other cases, however, it cannot be that the tubercle bacilli directly reach the organ which seems to be first affected. This is true of the so-called tuberculosis of serous membranes, of the tuberculosis of lymph-glands, of bones and joints, of the brain and of the genito-urinary tract. Investigators have yet to determine the paths by which the tubercle bacilli reach the respective organs in these cases. The glance which we have just taken at the organs most frequently attacked by tuberculosis shows the great clinical variety of tuberculous diseases viewed from this aspect.

Another reason for the great variation in the course of tuberculosis is found in the fact that the extension of the local tubercular process varies very greatly as regards time. Tuberculosis in one case may produce the most extensive destruction in both lungs in a few months or even weeks, and in another case it may remain almost quiescent for years, or advance only very slowly. We do not know fully on what these differences depend, but much is certainly due to the hygienic influences under which the patient lives. In the last analysis, however, we are often led to think of individual differences of constitution, which now check and now favor the rapid extension of the disease.

This predisposition is, in most cases, congenital, but sometimes acquired. This is particularly true of alcoholic subjects, who often evince a feeble power of resistance to tuberculosis, although originally of vigorous constitution, so that the rapidly spreading or "galloping" forms of tuberculosis are particularly common in drunkards.

A third and final reason for the differences in the course of tuberculous infection is the manner of the further extension of the tuberculous poison in the body. As we shall see in the description of tuberculosis in single organs, there are different ways in which tuberculosis may pass from one organ to another. Many contingencies are involved here, and we can easily comprehend how greatly the whole clinical course of the disease must be modified by the rapidity and the degree in which individual organs are affected.

After these preliminary remarks we will pass on to the description of the clinical course of pulmonary tuberculosis.

The onset of pulmonary tuberculosis is quite slow and gradual in the majority of cases. The patient can give only an approximate idea of the time when he began to be ill. The symptoms are referred directly to the respiratory organs. The cough and its attendant expectoration are the chief

things which affect him. Moreover, there is often pain in the chest, either the pleuritic stasis, or pain in the sternal region, or pain between the shoulder blades. Some physicians are inclined to refer these backaches of incipient tuberculosis to tuberculous disease of the bronchial lymph-nodes (*vide supra*). The patient is also apt to complain of shortness of breath, especially on exertion.

Besides these symptoms, which point pretty directly to disease of the respiratory organs, there are often quite striking general symptoms. The patient's emaciation is especially noticeable. This may be partly, though not wholly, explained by his loss of appetite. Besides the emaciation there is a steadily increasing pallor of the skin. The patient also shows a general dullness, weakness, and disinclination to work. There is not infrequently a slight rise of temperature in the first stages of the disease, which causes subjective feelings of chilliness and fever. Severe night-sweats may also be noticed early. The pulse rate is almost invariably accelerated, even in the absence of fever.

Any such constitutional disturbance should lead the physician not to regard the mild thoracic symptoms, which are also present, as insignificant, but to think of the possibility of incipient tuberculosis. It is very important to remember that the pulmonary symptoms may be entirely subordinate to the general symptoms mentioned, and that the patient himself is apt to pay little or no attention to them. Incipient phthisis is therefore frequently diagnosticated as simple "chlorosis" or "gastric catarrh" for a long time, and is treated as such. An early and careful physical examination of the lungs and of the expectoration is the only protection against such an error.

Both the pulmonary and the general symptoms assume significance, if we have to do with a patient in whom we suspect a "tuberculous predisposition." We very often meet persons in whose family, either in the parents or the brothers and sisters, several cases of phthisis have occurred. They are persons who are always pale and weak, and who have previously shown a special liability to catarrh and other diseases of the respiratory organs—e. g., pneumonia. They have perhaps had diseases which our present theories bring into direct relation with tuberculous infection. We refer to those quite frequent cases of pulmonary tuberculosis in individuals who have previously suffered from "scrofulous diseases," such as chronic swelling of the lymph-glands, chronic affections of the eye or ear, or fungous diseases of the bones and joints, pleurisy, etc.

Although the first symptoms of pulmonary tuberculosis often develop in those who were not quite well before, this is true in only a part of the cases. We often see precisely the same symptoms, both the pulmonary and the general, occurring in persons who previously seemed quite well and strong. No constitution is perfectly protected against the disease. We have even seen the herculean athlete of a circus die of phthisis.

In distinction from the slow and gradual method of the development of tuberculosis which has just been described, the first symptoms in other cases may be more sudden. A definite exposure is often given as a cause, after which the first symptoms of the disease have speedily developed. It goes without saying that we must consider these harmful influences—a chilling of the body, a cold draught, overexertion, or marked mental excitement—at

most, as exciting causes. When pulmonary tuberculosis begins somewhat abruptly, either the initial symptoms are from the start directly referable to the respiratory organs (cough, thoracic pain, dyspnœa), or the chest symptoms may be obscured by the severity of the constitutional disturbance. Thus we recall a number of cases in which young persons fell sick rather suddenly with somewhat severe, febrile constitutional disturbance. At first no cause of the fever could be found, so that the diagnosis was doubtful, or was supposed to be typhoid or some other disease. Then after a time thoracic symptoms developed and pulmonary tuberculosis could be recognized. In most cases the illness would take a rather rapid course. That form of pulmonary tuberculosis which is termed "pneumonic" (*vide infra*) also exhibits a decidedly acute onset.

Those cases of pulmonary tuberculosis which begin with hemoptysis are of special practical importance. Of course, it must be understood that the tubercular process in the lung has existed for some time before the blood appears. But in the midst of apparent health, or after some slight constitutional disturbance, comes a cough with bloody expectoration. Very often the further symptoms of pulmonary tuberculosis follow directly upon this initial hemoptysis (*vide infra*).

In conclusion, those cases are to be mentioned in which the first signs of tuberculosis appear not in the lungs but in the larynx. The full description of these cases has already been given in the chapter on laryngeal tuberculosis.

The further course of pulmonary tuberculosis may vary so much that it is impossible to give a complete enumeration of all the possibilities.

In some cases it advances rapidly. We can make out the extension of the disease objectively, almost from week to week. At first the apex of one lung alone is attacked, soon after the lower lobe of the same lung, then the other lung, either at the apex first or in the lower part. Besides the pulmonary symptoms, there is quite a high fever, rapidly increasing emaciation, and general loss of strength. Death ensues in a few months. We term such cases florid phthisis, or "galloping consumption."

In other cases, however, the disease has a remarkably chronic course. Its onset is very gradual, or else, after rather an acute onset, there is a comparative cessation of all symptoms. The thoracic symptoms do not disappear, but they are only trifling, and do not disturb the patient. Physical examination of the lungs does not show any extension of the process for months. The fever which accompanies it is slight, if there be any. The patient remains quite well nourished, but in some cases there is a good deal of weakness. He feels better and worse by turns, his condition being greatly influenced by the care and nursing he receives.

Unilateral contracting phthisis especially has this comparatively favorable course (*vide supra*). The affection remains confined to one lung for a long time. The occurrence of contraction shows the slight tendency of the tubercular process to advance, and with satisfactory care the patient may remain quite well for years.

In cases, too, which have had severe symptoms for a long time, a temporary standstill of the affection may take place, or an actual improvement in all the symptoms. At other times, in cases which have made no advance for a long time, the symptoms suddenly grow worse.

There are all possible varieties between the extremes of florid phthisis and the very chronic cases which last for years and decades. If we recall the further modifications which the course of the disease may assume if complications arise, we can appreciate the manifold character of the clinical picture of phthisis.

Most cases with definite signs of somewhat extensive disease terminate fatally. Death ensues, either with the signs of general exhaustion, or as a result of the final failure of respiration; or it is due to the occurrence of complications, such as tuberculous meningitis, miliary tuberculosis, pulmonary hemorrhage, or pneumothorax. Yet, on the other hand, if the tuberculosis is not extensive, the disease may terminate in complete recovery. To say how frequent recovery is, is difficult, for it is probable that many insignificant cases escape diagnosis. Furthermore, a distinction should be drawn between recovery from a standpoint of pathological anatomy, meaning complete cicatrization, with disappearance of all tubercular new growth, and recovery from a clinical standpoint [arrest], meaning disappearance of all symptoms. Often apparent recovery proves deceptive.

SPECIAL SYMPTOMS AND COMPLICATIONS

1. Lung Symptoms.—*Pain in the Chest.*—Extensive destruction of the lungs may occur without any feeling of pain. Many cases of phthisis are painless throughout their course. In other cases, however, the patient's chief complaint is of severe pains in the side or in the front of the chest. These are probably always due to coexisting affections of the pleura, such as pleurisy, or pleuritic adhesions. In patients who suffer from severe cough, pains sometimes arise in the abdominal muscles and at the insertion of the diaphragm, due to the excessive muscular contraction. It has been above pointed out that the diseased lymph-nodes at the roots of the lungs may, in some cases, be the cause for the spontaneous backache, or for the tenderness on pressure.

Cough.—In the majority of instances cough is one of the most distressing symptoms of phthisis, but its severity varies very much in different individuals, and at different times in the same patient. We sometimes see cases, particularly in senile, insensitive individuals, in which, in spite of advancing phthisis, cough is remarkably slight, or entirely absent. In cases with severe cough, it is often worse at night, but paroxysms of coughing of long duration are also apt to come on in the morning or evening hours, which are painful and very distressing, and exhausting for the patient. The cough is usually associated with a more or less abundant expectoration, but sometimes there is chiefly a dry cough. The cough becomes very severe if the tuberculous affection attacks the larynx and trachea (see laryngeal tuberculosis).

Expectoration.—The amount of expectoration differs very much in different cases. It is most abundant when there is extensive formation of cavities in the lungs. In such cases it is often evacuated in the morning by persistent coughing. The consistence of the great part of the sputum is mucopurulent, and it differs little from that of simple bronchitis; in fact, a large part of the phthisical expectoration comes from the catarrhal inflammation of the bronchial mucous membrane. Usually, however, the amount of mucus

as compared with the amount of pus is less in phthisical sputum than in that of simple bronchitis. The sputum is therefore less viscid and more fluid. The amount of mucus in the expectoration is also apt to be greater in chronic bronchitis than in tuberculosis, although mucus is seen in the latter. The expectoration which comes from cavities is almost pure pus, with only a slight amount of serum and mucus intermixed. Such sputum consists often of large separate masses described as nummular, or coin-shaped. If received in water, the uneven, rough surface of these masses is often evident, and suggests their formation in the irregular pulmonary cavities.

The admixture of blood with the sputum is of great diagnostic and practical importance. Since no other disease so often gives rise to the presence of blood in the expectoration, coughing of blood (hemoptysis) is almost synonymous with consumption among the laity. Little streaks of blood in the expectoration are quite frequent. They have no great significance, but, of course, they may sometimes be the precursors of severe hemorrhages. Profuse hemoptysis takes place when the wall of a little pulmonary vessel—almost always a branch of the pulmonary artery—is infiltrated, destroyed, and finally eroded, by the tubercular new growth. The reason why hemoptysis is not more frequent is because the contents of the vessels usually undergo thrombosis. Severe hemorrhages very often have their origin in little aneurisms of the branches of the pulmonary artery, which penetrate into the interior of the cavities. In the cases of fatal hemoptysis we frequently succeed in finding the little aneurism and its point of rupture.

Pulmonary hemorrhage occurs in all stages of phthisis. The initial hemoptysis has already been mentioned. This may be followed by other symptoms of pulmonary tuberculosis, or the hemoptysis may cease without any immediate sequels. Pulmonary hemorrhage may also occur at any time in the further progress of the disease. The amount of blood raised is variable. There may be one or more tablespoonfuls, or one or more pints. The blood is bright red, usually quite frothy, but little clotted, and in part mixed with other constituents of the sputum. After the first well-marked hemoptysis, the expectoration usually is mixed with blood for several days. Again, there may be repeated hemorrhages in a short time. Sometimes the hemoptysis begins abruptly, not infrequently at night, without any occasion. In other cases, the hemorrhage is referable to some distinct cause, such as bodily exertion, a violent paroxysm of coughing, straining at stool, mental excitement, and the like. Many cases of phthisis are characterized by a special tendency to hemorrhage, while in many others hemoptysis never occurs. Severe hemoptyses are, of course, always an undesirable and dangerous complication, since they weaken the patient very much, and also depress his spirits. Many patients maintain their peculiar careless indifference, which is almost characteristic of the disease, despite the spitting of blood. The hemoptysis may sometimes be the direct cause of death, but, as a rule, the patients survive it. We have no better means of determining with certainty the influence which hemoptysis has upon the progress of tuberculosis than by the bodily temperature. If there was no fever before, and the hemoptysis also has an afebrile course, or only a very brief rise of temperature, then we may hope in general that the patient may fully recover from the hemorrhage and be as well as before. If, however, the hemoptysis is followed by persistent fever, or if the fever which may have

previously existed becomes higher and more persistent, we have every reason to suppose that the tubercular process is making more rapid advances subsequently to the hemoptysis. We here append a temperature chart (Fig. 50) illustrating the temporary influence of a hemoptysis upon the temperature in

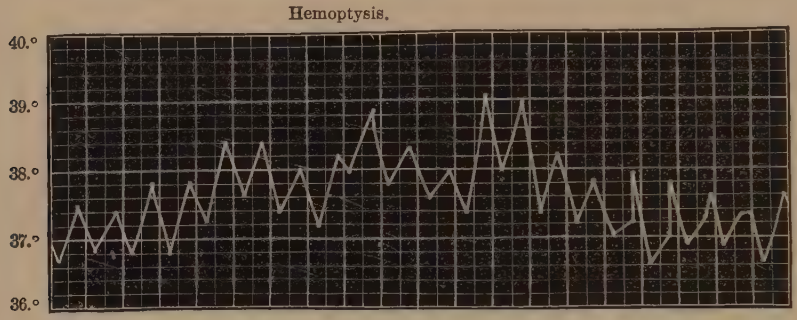

FIG. 50.—Influence of a pulmonary hemorrhage upon the bodily temperature. (Erlangen Medical Clinic.)

a stationary afebrile case of pulmonary tuberculosis. The fact that the fever preceded the hemorrhage by a few days is probably due to the tuberculous arteritis, because of which material capable of exciting fever entered the circulation before the process had caused complete destruction and bursting of the arterial wall, and consequent pulmonary hemorrhage.

A purulent sputum intimately mixed with blood is quite frequent and characteristic in many cases of phthisis with extensive formation of cavities. This is formed in the cavities from the mixture of the purulent secretion with little capillary hemorrhages. In this way the sputum, which is often nummular, assumes a greasy character and a reddish-brown or chocolate color.

If fetid or gangrenous processes develop in the lungs, the sputum becomes fetid. In some cases we see temporarily in phthisis the characteristic sputum of croupous pneumonia, which comes from portions of the lung attacked with secondary pneumonia.

Microscopic examination of the sputum may show—besides the ordinary morphological elements, such as pus corpuscles, red blood corpuscles, and epithelium—two constituents which are of decided diagnostic importance: elastic fibers and tubercle bacilli.

The demonstration of elastic fibers in the expectoration permits us to decide with certainty that there is a destructive process in the lungs, and thus it usually is direct proof of tuberculosis. Elastic fibers are also found in pulmonary gangrene, and in cases of pulmonary abscess, as well as in tuberculosis, but these diseases are easily recognized by the other peculiarities of the sputum. The search for elastic fibers in the expectoration of tuberculous patients demands a certain amount of practice. We are most sure to find them if we look in the sputum, when it is spread out, for little lentiform particles, which can easily be discerned with the naked eye. These consist of necrotic shreds of tissue torn off from the walls of cavities. If we press one of these "kernels" under a cover-glass we find, in the midst of the granular detritus, beautifully twisted elastic fibers, which often have quite a definite alveolar arrangement (see Fig. 51). The elastic tissue is the only one spared

in the general destruction. There is a special method of looking for elastic fibers. The sputum is boiled in sodium hydrate, diluted with water, and we look for elastic fibers in the precipitate which then forms. We are never justified, however, in deciding that pulmonary tuberculosis is absent because we do not find elastic fibers in the sputum. Their presence alone has real diagnostic significance.

FIG. 51.—Elastic fibers from the sputum of a case of pulmonary tuberculosis.

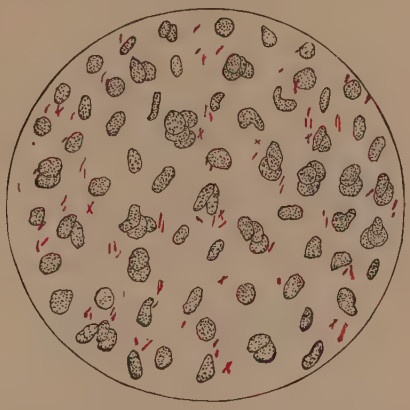

FIG. 52.—Tubercle bacilli in the sputum.

The discovery of tubercle bacilli in the expectoration of phthisical patients is of much greater importance, and often this alone is decisive (see Fig. 52). They were first demonstrated by Koch, but Ehrlich devised the first simple method for their discovery, which can be easily employed by any physician.

The simplest method, and the one which we now employ almost exclusively for staining the bacilli, is as follows: Some of the sputum is rubbed between two cover-glasses, which are then slowly separated, leaving upon each cover-glass a very thin layer of sputum. In order to fasten this sputum upon the cover-glass, the latter is passed three times slowly through a gas flame. The cover-glass is now held with a pair of forceps and covered with the following staining fluid (carbol-fuchsin solution), which was first proposed by Ziehl and Neelsen: Distilled water, 100 parts; crystals of carbolic acid, 5 parts; fuchsin, 1 part; mix, filter, and add alcohol, 10 parts. This staining solution upon the cover-glass is heated to boiling for a short time, and then the staining is completed. The cover-glass is now washed, first in absolute alcohol, then in distilled water, then placed for about two minutes in a solution of 2 parts of methyl blue in 100 parts of a twenty-five-per-cent solution of sulphuric acid. The acid bleaches out the diffused fuchsin stain, leaving the tubercle bacilli still colored, while the pus corpuscles are colored blue. The preparation is now rinsed in water, dried between two pieces of filter paper, and examined in Canada balsam. The whole process, when one has had a certain degree of experience, requires four or five minutes. The number of bacilli in different cases, and at different times in the same case, varies considerably. The more abundant the bacilli, the more reason have we to suppose there is a rapidly advancing process of ulceration, but of course we

can never draw a broad conclusion as to the extent of the tuberculous disease from the number of bacilli in the sputum. Our only information on this point must come from the physical examination, and other clinical observations. On the other hand, the diagnostic significance of the demonstration of bacilli with regard to the recognition of a pulmonary tuberculosis cannot be overrated. A positive result is absolutely decisive, and the diagnosis of tuberculosis of the lungs ought certainly never to be made unless the bacilli have been demonstrated. Very frequently bacilli may be found in the sputum in early cases, at a time when no other certain sign of tuberculosis can be detected. On the other hand, we scarcely need to point out that the physician should be cautious in the interpretation of negative results. In all suspicious cases we must repeat the examination of the sputum over and over again.

Dyspnœa.—Many patients hardly ever complain of their breathing in spite of extensive destruction in the lungs. A patient who is much emaciated manifestly needs little oxygen, and the increased frequency of respiration, which is almost constant, can satisfy his needs. If there is a greater demand upon the respiration a subjective feeling of dyspnœa may, of course, very readily occur, especially on a slight bodily exertion. In many cases, however, the patient complains of a difficulty in breathing even when quiet, especially if pleuritic pains or adhesions between the surfaces of the pleura prevent him from taking a deep breath; and in the final stages the dyspnœa may be extreme.

2. **Symptoms on Physical Examination.**[1]—In many cases inspection gives us that general impression of the patient which we term the "phthisical habit." The special signs of this are as follows: A slender but often quite a tall frame, weak muscular development, a thin layer of fat, a pale and perhaps very delicate skin with a bluish translucence, sometimes a circumscribed "hectic" flush in the cheeks, a long and slender neck, a long and flat thorax, small, thin hands, etc. Of course this characteristic constitutional habit is not equally well marked in all cases. The inspection of the thorax is of special value. The phthisical or "paralytic" thorax is generally noticeable from its length, but it is narrow and flat. Unusual width of single intercostal spaces, and acuteness of the epigastric angle, are associated with a long thorax. The sternum is also long and narrow, and the sternal angle—Louis' angle—is sometimes particularly prominent. The supraclavicular and infraclavicular fossæ are sunken, the neck is wasted, and the shoulder blades stand out from the thorax. On comparing the two halves of the thorax we very often observe a distinct drawing in and flattening (contraction) on the side most affected. This change is most frequently in the upper and anterior portions of the chest, but it is not rare even in the posterior and lower.

The paralytic form of thorax is very often seen in phthisis, but it may be entirely absent.

The respiration is usually somewhat accelerated, and sometimes quite markedly so in women with disease of the apices. The feminine type of high thoracic breathing is largely changed to low thoracic or diaphragmatic breathing. A unilateral impairment of respiration is of greater importance; in

[1] The significance of the X-ray examination will be considered in the section dealing with the diagnosis of pulmonary tuberculosis.

such a case the apex in front or even the whole side, if there be phthisis of the lower lobe, lags in inspiration. The respiration is sometimes irregular, especially if there be pleuritic pains.

The results of percussion are, of course, entirely dependent upon the sort of anatomical changes in the lungs, and hence differ very greatly in different cases. Since the phthisical process begins in the apices in the majority of cases, our chief attention is turned to the condition of the upper portions of the lungs on percussion. Slight changes in percussion may wholly escape discovery. Only when the air contained in the lung tissue in the part affected is replaced to a certain degree by the tubercular infiltration does the percussion note become dull. Unilateral dullness at the apex is therefore one of the most frequent physical signs of phthisis. We can make it out most plainly in the upper anterior intercostal spaces first, and in incipient cases often in the supraclavicular fossæ only, but it is also observed sometimes in the back in the suprascapular fossæ.

As the infiltration advances the dullness becomes more extensive. For the accurate determination of the limits of dullness in the upper portions of the lungs, we recommend that percussion should be practiced in such a way as to proceed from the normal resonance of the lower portions upward toward the affected area. This method is particularly useful for the back of the chest. If light percussion is practiced from below upward on both sides of the vertebral column of a normal chest, the percussion note will be of the approximately same clearness and pitch up to the apices. If, on the other hand, as is the rule in tuberculosis, we are dealing with disease in the upper portion of the lungs and normal lower lobes, a decided change in the percussion note will be heard either at the level of the root of the lungs or when the upper lobes are reached. The note is dull, or its pitch is changed, becomes tympanitic, etc. This method of percussion for the demonstration of beginning apical dullness appears more advantageous to me than the method usually employed, of comparison of the percussion notes on the two sides. With simultaneous disease of both apices, slight dullness may be more easily overlooked with the methods of percussion usually employed. Within certain limitations the vertical method of percussion from below upward also gives the most trustworthy information as to the extent of the lesion in cases of advanced tuberculosis. In the percussion of the apices, Krönig lays special stress upon the diagnostication of possible retraction of the apices, by the fact that, with the patient in the horizontal posture, the width of pulmonary resonance is diminished in the supraclavicular fossæ in front, or over the apices, posteriorly. This method sometimes gives very good results, but, in my experience, it is of less practical value than the direct demonstration of apical dullness. Often the dullness takes a tympanitic quality as a result of diminished tension in the lung, or more or less pulmonary retraction. Changes in tension may render the resonance, in beginning tuberculosis, deeply tympanitic without any accompanying dullness.

The formation of cavities in tuberculosis has a great influence on the percussion note. The dullness on percussion may thus become decidedly less, the degree of resonance depending, of course, upon the fullness of the cavity and the character of the surrounding tissue. We often find a decided tympanitic resonance or a combination of dullness and tympany over a cavity. The dif-

ferent modifications of the percussion note in cavities are given below. The "cracked-pot resonance," or buckram sound, is met with in percussing over cavities, but we also find it in many other pathological conditions, and in children not infrequently when the lung is normal.

Auscultation also gives no special pathognomonic signs of phthisis. Varying with the character and extent of the tuberculous changes, abnormal respiratory sounds and adventitious sounds are heard in place of the normal vesicular murmur. With slighter changes the vesicular breathing is merely modified; it seems remarkably diminished or interrupted ["cog-wheel"], or sometimes exaggerated, with prolonged expiration. When the infiltration of the lungs increases, we find bronchial respiration in place of the vesicular breathing; but, on the other hand, the formation of a cavity is a frequent cause of bronchial respiration.

Various sorts of moist râles are among the most frequent and diagnostically important of the auscultatory signs of phthisis. They are caused by the collection of secretion in the bronchi, or in pulmonary cavities. The more abundant and liquid the secretion is which is set in motion by the air current which traverses it, the more abundant and moister the râles. The larger the space is in which they develop, the coarser they are. Besides the true moist râles, there are also dry bronchitic râles (sibilant or sonorous) occasionally to be heard in portions of the tuberculous lung. In general, we may say that the extent of the tuberculous disease in the lungs can be determined by no symptom so surely as by the auscultatory signs relating to the respiration, and the adventitious sounds which may be present.

Physical Diagnosis of Incipient Phthisis.—The importance of the diagnosis of early phthisis is so great that we will briefly sketch its most important physical signs. Now that the examination of the sputum for tubercle bacilli plays by far the most prominent part in the recognition of incipient tuberculosis, and is the only decisive evidence, the physical signs of this condition have lost much of their former importance. Despite this, the determination of the seat and extent of the early process is extremely desirable. We will mention the following symptoms: 1. Constant and evident diminution of the respiratory murmur at one apex, especially if it is associated with marked deficiency of the respiratory movement on the affected side. In some cases the respiratory murmur on the diseased side is not weaker, but it has a more indefinite and harsher character; or, again, it may be rude, sharp, and "puerile." 2. Markedly interrupted respiration at one apex. 3. A prolonged expiratory murmur, which has a harsh character. 4. The discovery of dry rhonchi or moist râles at one apex is most important, since we know by experience that "apex catarrhs" are, almost invariably, tuberculous. 5. Definite dullness, apparent on repeated examinations, or tympanitic dullness or tympany at one apex. 6. Evident contraction at one apex, as revealed by inspection or percussion above the clavicles. 7. Some authors lay stress upon a systolic murmur in the subclavian artery, especially loud on expiration. This is said to occur in the beginning of phthisis, if the caliber of the vessel is narrowed by processes of contraction in the neighboring apex. The symptom has no practical significance, however, since similar vascular murmurs are occasionally heard over normal apices.

The chief rule in the diagnosis of incipient phthisis must be held to be

this—not to give a definite opinion until repeated examinations have been made. The other portions of the lungs are to be carefully examined as well as the apices, since in not very rare cases tuberculosis may begin in the lower lobes. We must always consider other circumstances (heredity, constitution, general symptoms of the disease, fever, expectoration, etc.), as well as the physical signs presented by the patient. These as well as the specific, biological reactions and the extremely valuable aid to diagnosis rendered by the X-rays, will be considered below.

Diagnosis of Cavities.—The positive diagnosis of pulmonary cavities by means of physical examination is often very difficult. Anyone who will compare the discoveries at autopsies with the results of previous physical examination of consumptives will be in a position to confirm the truth of this statement. We may mention as the chief symptoms of a cavity: 1. Loud bronchial respiration, perhaps of an amphoric character, in places where the percussion note is only slightly or not at all dull, but perhaps tympanitic. Such a condition means that the bronchial respiration is not due to an infiltration of lung tissue. Bronchial respiration, however, may, of course, be heard over cavities which are surrounded by thickened lung tissue, and hence give dullness on percussion. Pure amphoric respiration is very characteristic of a cavity, particularly if there is a clear, metallic quality to the sound; but this sort of respiratory murmur is heard only when the cavity is comparatively large, of regular shape, and with smooth walls. Under these circumstances the moist râles, if there are any, also have a clearly metallic sound (tinkle), and the percussion resonance may also be distinctly metallic. This last should be demonstrated by rod percussion with simultaneous auscultation. In many cases of this sort the differential diagnosis, between a large cavity and a sacculated pneumothorax (see page 364), is very difficult. 2. The so-called metamorphosing respiration, which begins as vesicular and suddenly becomes bronchial, is heard especially over cavities, and hence has a diagnostic value. 3. The different kinds of "changes in the percussion note" over cavities are important signs. The most frequent and of greatest practical importance is Wintrich's change of note. This is when the tympanitic resonance, which is obtained over the cavity, becomes, on opening the mouth, more decidedly tympanitic, louder, and especially much higher. The respiratory change of pitch of Friedreich usually consists of a higher pitch on inspiration, but here there are numerous variations. Gerhardt's change of pitch (Weil) consists in a change of the tympanitic resonance when the patient changes his position, the pitch usually being higher when the patient sits up than when he is lying down. 4. Loud, bubbling râles are one of the most frequent signs of a cavity. They are definite indications of the occurrence of râles in a larger space than is normally present in the apices of the lungs. If we have coarse and metallic râles in the apices of the lung, there is considerable probability of the existence of a cavity, inasmuch as the normal apices cannot produce such sounds. 5. Finally, we would point out that large cavities not infrequently appear in X-ray pictures in the form of distinct, sharply circumscribed, lighter areas.

In addition to the physical signs, the nature of the sputum must be considered. As a rule, a profuse, purulent clumpy ("nummular") expectoration indicates cavity formation. In febrile cases of phthisis with remarkably little expectoration, large cavities can, as a rule, be excluded.

Contraction of the Lungs [Fibroid Phthisis].—Unilateral contraction of the lungs, much more frequent on the left than on the right, is a form of tuberculosis which is made apparent both by special physical signs and also by certain clinical peculiarities. It is usually recognized at once by inspection of the thorax, one side of the thorax being remarkably retracted. The upper anterior portions of the thorax, and, in all cases of a high degree of disease, the lower lateral and posterior portions, are much less expanded than the corresponding parts on the other healthy side. The fossæ and intercostal spaces on the affected side are deeper, the shoulder blade is drawn nearer the vertebral column, and the latter is even sometimes drawn over to the contracted side (scoliosis). The resonance is diminished to a greater or less degree over the affected side, which either lags behind or remains almost wholly at rest on respiration. The respiratory murmur is quite loud and bronchial; and we also hear many râles, which are usually bubbling. Exceptionally the râles are few and the respiratory murmur feeble and indistinct. Anatomically, we have to do with a marked process of contraction of the interstitial connective tissue in the lungs, which is almost always associated with extensive formation of cavities, partly of an ulcerative, partly of a bronchiectatic character. The pleura is almost invariably involved in the process, but with the exception of the cases that have begun with a pleurisy with effusion this is only secondary. The pleura is also thickened and contracted. If the pleuritic thickening is marked, the respiratory murmur and the vocal fremitus are decidedly weakened.

The influence of the contraction on the neighboring organs is very decided, and usually it is easy to discover. The heart especially, whose external pericardium is in most cases very adherent to the pleura, is drawn well over to the side of the contraction. The apex beat and the cardiac dullness are correspondingly displaced. With left-sided contraction the heart may be drawn over to the line of the left axilla, and with right-sided contraction it may be drawn to the median line, or even to the right of the sternum. With contraction of the left upper lobe the anterior surface of the heart comes into immediate contact with the anterior chest wall over a larger area than normal. We therefore see the motions of the heart over an abnormal extent, and we can often feel very plainly in the second left intercostal space the pulsation and the closure of the valves in diastole of the pulmonary artery. The upward traction of the diaphragm may be recognized by the position of the liver, or, in left-sided contraction, by the increase of the "semilunar" tympanitic space on the left. We usually find the sound lung on the other side quite emphysematous, as shown by the downward displacement of the lower boundary of the lung, and also by the drawing over of the anterior median edge of the lung to the contracted side. In a part of the cases we can make out by percussion the development of consecutive dilatation and hypertrophy of the right ventricle.

These are the chief physical signs of the so-called unilateral form of chronic pulmonary contraction. We would add here a few clinical remarks. These cases often, but of course not always, run a very chronic course, lasting for years. The general condition and the nutrition of the patient may thus remain comparatively undisturbed for a long time. The patient looks somewhat pale and cyanotic, yet he is so well nourished as to present a very marked contrast to the appearance of the ordinary cases of phthisis. The appetite

remains good, the fever is entirely absent, or else a slight degree of fever may be at times discovered by careful examination. The cough and expectoration, too, though often quite troublesome, are at other times very slight, especially when the patient has good care and nourishment. We need not wonder, then, that formerly many physicians did not consider that these cases had anything to do with phthisis—"consumption"; and yet we are convinced by many clinical and anatomical observations that, ætiologically, they are in by far the greatest part tuberculous. They represent a very slow form of tuberculosis, which has time to develop interstitial processes which lead to contraction—that is, to local healing. If such cases come to autopsy, their tuberculous character is usually definitely confirmed. We find undoubted tuberculous lesions in the other lung and also in the remaining organs—e. g., the intestines. Sudden changes for the worse are always possible in "pulmonary contraction," even in those cases which seem favorable; the other lung may become highly tuberculous, a miliary tuberculosis or a tuberculous meningitis may develop, etc. On the whole, however, the slow course of this form of chronic tuberculosis is characteristic and of great practical significance, and its prognosis is therefore comparatively favorable.

We cannot deny that a nontuberculous unilateral contraction of the lung may occur. As a result of bronchitis (particularly in cases due to the inhalation of dust), and also after pleurisy, processes of contraction develop, which are associated with the formation of bronchiectasis, and certainly have nothing to do with tuberculosis. In rare cases also croupous pneumonia is followed by unilateral contraction of the lung; and finally there is a rare and by no means satisfactorily investigated form of unilateral chronic interstitial pneumonia, with contraction, often associated with the formation of bronchiectasis. The differential diagnosis of these conditions from tuberculous contraction rests in part upon the clinical history, which should be carefully taken, but mainly upon the absence of tubercle bacilli in the expectoration.

In conclusion, we must mention that there are very many transitional forms between pulmonary contraction and the other varieties of pulmonary tuberculosis. We find more or less extensive processes of contraction in one apex in most cases of phthisis.

Disseminated Pulmonary Tuberculosis.—There is a form of pulmonary tuberculosis which it is very hard to make out on physical examination. In this we have to do with numerous peribronchial nodules disseminated over the whole lung. As there is still a good deal of normal tissue, containing air, between these nodules, percussion affords no dullness, and auscultation gives at most diffuse rhonchi; hence this form is often confused with chronic bronchitis or pulmonary emphysema. The diagnosis can seldom be made from the physical signs, but only from the other symptoms, such as fever, emaciation, striking pallor of the skin, and the sputum. X-ray examination may give important information as to the condition of the lungs.

The disseminated form of phthisis sometimes runs a chronic course, but usually it is quite rapid. It occurs in elderly people, and also in children. Many forms of "disseminated, coarse granular" tuberculosis are transitional forms between this and genuine acute miliary tuberculosis (*q. v.*).

Pneumonic Form of Pulmonary Tuberculosis.—A special clinical interest attaches to that form of pulmonary tuberculosis termed pneumonic,

which we have already briefly mentioned. The disease begins in a decidedly acute manner with dyspnœa, frequent cough, and pain in the side. Sometimes we observe even a distinct initial chill. The expectoration is scanty, viscid, mucous, and often reddish or hemorrhagic. It sometimes also has a peculiar greenish color. Even after a few days of illness we find, on examination, the well-marked signs of lobar infiltration. There is first pneumonic resonance, then a dull tympany, with fine moist râles and bronchial breathing. In most cases, but not all, a lower lobe is involved. The disease is almost invariably regarded at first as a croupous pneumonia, but the expected crisis does not appear. The fever remains high, the infiltration does not undergo resolution, the râles become coarser, the patient looks pale and wretched. Now, suspicion of tuberculosis being aroused, the sputum is carefully examined, and soon, although perhaps not at the first examination, tubercle bacilli are found. Nearly all cases of this sort take a rapid and unfavorable course. They may be called galloping consumption. Upon autopsy we find diffuse tuberculous infiltration, and usually incipient cavity formation in numerous places. Other portions of the lungs than that first and most violently attacked ordinarily present considerable tuberculous change. In these instances it is evident that we have to do with an acute infection of a large portion of the lung with tubercle bacilli, which of themselves exert a powerful inflammatory action, with unusual virulence. In some cases there may also be a mixed infection, due to the pneumococcus.

We have seen a few cases in which the pneumonic form of tuberculosis followed a severe hemoptysis. In these instances the aspirated blood probably led to the rapid spread of the infection.

3. **General Symptoms in Pulmonary Tuberculosis.**—In the description of the general course of pulmonary tuberculosis we have already mentioned the value of the constitutional symptoms in diagnosis and prognosis.

Fever.—Only a few cases of phthisis run their course entirely without fever, but it may be absent for a time, even for weeks and months; and indeed, in cases with a very slow and favorable course (e. g., where there is unilateral contraction), there may be no fever at all for years. The more carefully we measure the temperature the more often shall we find slight elevations of the same, even when the patient is feeling well. In normal individuals the axillary temperature hardly ever rises above 98.6° to 99° F. (37° to 37.2° C.), and generally it is somewhat lower than 98.6° F. (37° C.). A persistent temperature above 98.6° F. (37° C.), and especially frequent elevations to 99.5° or 100.4° F. (37.5° to 38° C.) should be considered as decidedly pathological. Such slight elevations of temperature (so-called subfebrile state) often persist for a long time, or at least occur occasionally in numerous cases of tuberculosis (see Fig. 53). Ele-

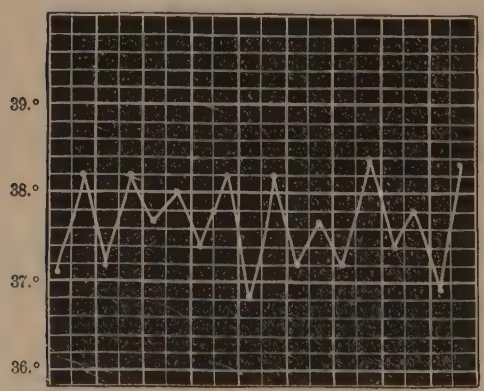

Fig. 53.—Subfebrile state in chronic pulmonary tuberculosis.

vations of temperature to 100.4° or 101° F. (38° to 38.5° C.), especially when observed in the evening, indicate increased activity of the tubercular process.

Very often, particularly in the more rapid cases, there is considerable elevation of temperature, and the varieties and course of the fever are more or less characteristic. We should first mention the "hectic fever" (see Fig. 54) which is often observed. For months the temperature chart may present a uniform appearance with a morning temperature approaching or reaching the normal, while there is a regular elevation every evening to 102° or 104° F. (39° to 40° C.). In general, the higher the evening exacerbations the more unfavorable the case. In some cases the temperature rises in the morning and drops in the evening. This is the so-called inverted type, and is usually an unfavorable symptom. In other cases the temperature chart is quite irregular; longer or shorter periods of persistent elevation alternate with periods when there is no fever. Particularly toward the end of the disease, as the bodily weakness increases, the previously regular, intermitting temperature grows irregular. At this time the intermissions often become more marked, and not infrequently collapse temperatures—95° to 93.2° F. (35° to 34° C.)—are observed. Again, the fever may for limited periods take on a more continuous character, probably in connection with exacerbations of the tubercular process. In some few cases with acute onset (*vide supra*) we have also seen, in the beginning of the disease, a tolerably high and approximately continuous fever, passing gradually into the ordinary *febris hectica*. The cause of the fever in pulmonary tuberculosis is not yet settled. The special question is whether the tubercular process causes the fever of itself (by production of toxins), or whether the fever is due to the secondary inflammatory processes, such as the absorption of septic and toxic material from the decomposing contents of the bronchi and the pulmonary cavities. The practical importance of the fever in pulmonary tuberculosis is very great. The emaciation and weakness of the patient are caused mainly by fever (*vide infra*), as are many subjective symptoms, including headache, chilliness, and perspiration. It is important to remember that in observations of the fever we possess one of the most positive means of forming an opinion as to the course of pulmonary tuberculosis. In cases which are either completely stationary or very chronic there is no fever at all. The subfebrile condition shows a slow, but still continuous, advance of the disease. Continuous hectic fever is an unfavorable sign, and indicates a comparatively rapid march of the disease—the more rapid, the higher the evening temperature. In the florid or galloping forms we find tolerably high fever, at times continuous and at times remittent. Subnormal temperatures are almost always of bad omen.

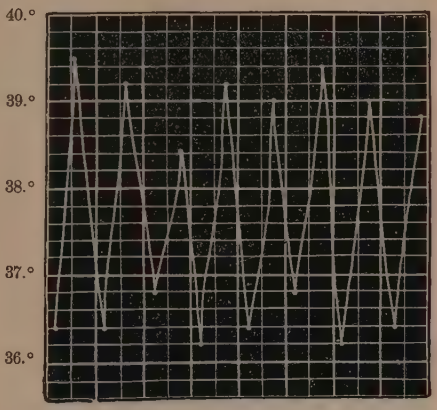

FIG. 54.—Hectic fever in chronic pulmonary tuberculosis.

All changes in the general course of the disease for better or worse stamp

themselves clearly on the temperature chart. The onset of complications and secondary disease is often first indicated by the thermometer. Whether an attack of hemoptysis passes over without permanent damage, or whether it is followed by an aggravation of the disease, can usually be told by the temperature sooner than in any other way (*vide supra,* page 290). Thus we see that the persistent and careful observation of the temperature in consumption deserves to rank as one of the most important means of estimating the status and course of the illness.

Emaciation.—The great emaciation of the patient is very striking in most cases of phthisis. The muscular system and the fatty tissue are affected in equal degree. The soft parts of the thorax are often especially involved. The emaciation is due in part to the patient's loss of appetite, and to the small amount of food which he takes in consequence thereof, but the chief cause lies in the persistent fever and the increased metamorphosis of tissue. Quite a high degree of emaciation, however, may appear in the beginning of the disease with no fever. This we are wont to ascribe to the "general illness," but the special cause of it is unknown. Under favorable external conditions phthisical patients may make quite a decided gain in weight, especially at the times when they are free from fever. In very chronic cases, which run their course from the first without fever, the nutrition of the patient may remain good for a long time. Toward the end of the disease emaciation sometimes reaches its highest degree, and many phthisical patients die "wasted to a skeleton" in the true sense of the word.

Anæmia—Color of the Skin.—In most cases anæmia appears in the course of the disease, to be recognized by the pale and sallow color of the skin and of the visible mucous membranes. The anæmia only rarely reaches that degree of peculiar waxy pallor that is found in idiopathic pernicious anæmia. If the pallor is extreme, however, there is usually some special reason, such as profuse hemoptysis or the complication of amyloid degeneration. The existence of the anæmia is also the reason why the phthisical patient does not look cyanotic in spite of the respiratory disturbance. In the more chronic forms, where the general nutrition suffers less, we often see a cyanotic coloring of the lips and cheeks. Sometimes the skin of phthisical patients assumes a dirty, dusky hue. We have already spoken of the circumscribed "hectic flush of the cheeks" seen with the fever.

General Weakness—Night-sweats—Nervous Disturbances.—We need not say that the general emaciation and anæmia are accompanied by a marked decline in the patient's strength. He finally becomes so helpless that he can scarcely move alone in the bed.

The tendency which very many patients have to severe night-sweats is universally acknowledged but not wholly explained. It may have some connection with the fall from the evening febrile temperature to the morning remission, and perhaps it is due to the greater accumulation of carbonic acid in the blood from the disturbance of respiration.

The disease has remarkably little influence upon the higher nervous functions, especially those of the mind. Most patients have a perfectly clear intellect to their latest breath. We all know the contented, hopeful, sanguine disposition of many patients, who do not recognize their own danger until the last stages of the disease. Occasionally the anæmia and the general dis-

turbance of the nutrition of the brain lead to mental alterations, such as confusion, distraction, or melancholic conditions.

We find, more frequently, disturbances in the peripheral nerves and muscles. Among these are neuralgic pains, and pains of an indefinite character, which have their seat in the legs, or sometimes in the arms, especially in the ulnar region and the sciatic nerve. These may be very distressing. Marked hyperæsthesia of the skin and deeper parts is also not uncommon. The cause of such disturbances is probably often to be looked for in the degenerative changes in the peripheral nerves (Vierordt and others). Well-marked multiple neuritis has been repeatedly observed in tuberculous patients (see Vol. II).

We very often see an increased reaction upon direct mechanical irritation in the emaciated muscles, and great liveliness of the so-called idiomuscular contractions, which is shown, for example, on percussing the pectoral muscles on the anterior wall of the chest. The phenomena grouped under the name of tendon reflexes are also much increased in phthisis.

4. **Symptoms and Complications on the Part of Other Organs.**—1. *Pleura.*—In pulmonary tuberculosis the pleura is also involved, as a rule. The affection is almost always the result of a direct extension of the process from the lung to the pleura. At the autopsy, we find in the pleura a few or many miliary tubercles, besides the simple inflammatory process—tuberculous pleurisy.

In many cases, in which we have to do only with an adhesive pleurisy and with pleuritic contraction, we can merely suspect the disease of the pleura, but it cannot be directly made out and differentiated clinically from the pulmonary affection. In other cases we can diagnosticate a dry pleurisy in phthisis from the occurrence of the pleuritic friction rub. The symptoms of pleurisy become more marked if there is a pleuritic effusion, which is usually readily discovered by a physical examination. The patient's pain and dyspnœa are usually much increased by such a complication. Besides a simple serofibrinous effusion we quite frequently find purulent and even hemorrhagic effusions in tuberculosis of the pleura.

The formation of pneumothorax is an important complication in the pleura in phthisis. It is due to the rupture of a superficial pulmonary cavity into the pleural cavity, and the entrance of air into the latter. The different forms of pneumothorax and its symptoms will be described under diseases of the pleura.

2. *Larynx, Trachea, and Pharynx—Buccal Cavity.*—The symptoms of laryngeal tuberculosis and their relation to pulmonary tuberculosis have already been given under diseases of the larynx (see page 178). We saw there that, although there is a primary laryngeal tuberculosis, most cases are secondary in their development to a pulmonary tuberculosis.

The same holds true in regard to the much rarer tuberculosis of the pharynx. In some cases this may be of primary origin, but it is usually a result of reinoculation with tuberculosis by means of the sputum, or of a direct extension of the tubercular process from the larynx to the pharynx. Tubercular ulcers of the pharynx are found most frequently on the soft palate, on the tonsils, on the root of the tongue, and on the boundary between the pharynx and the larynx; they are rare in other parts of the pharynx. In exceptional cases tuberculous affections are seen in the mouth—on the tongue.

The local discomforts which all these ulcers cause is usually very considerable. Disseminated miliary tubercles, too, have been repeatedly seen in the mucous membrane of the pharynx. Pronounced thrush formation sometimes develops in the pharynx of patients who are very ill.

3. *Stomach and Intestinal Canal—Peritoneum.*—Tubercular ulcers in the mucous membrane of the stomach are exceedingly rare, but we very often notice some symptoms on the part of the stomach. Loss of appetite is a particularly common symptom in phthisis. Vomiting occurs often in phthisical patients, especially when the larynx is involved. It is usually brought on by paroxysms of coughing. Less frequently the cause of the vomiting is gastric catarrh, occasioned by the irritation of the sputa which have been swallowed; but in some cases the gastric symptoms depend upon the general condition—e. g., the anæmia.

Although the tubercle bacilli swallowed with the sputum hardly ever infect the stomach, probably from the acid reaction of its contents, they very often attack the intestinal canal. In the majority of the cases of phthisis we find tubercular ulcers, either singly or in considerable numbers, in the vicinity of Bauhin's valves [the ileocæcal valve], in the lower part of the ileum, and the upper part of the large intestine.

Intestinal tuberculosis (*q. v.*) does not always cause very marked clinical symptoms, but, as a rule, we find diarrhea in patients with tubercular ulcers of the intestine. They may have three or four stools in the twenty-four hours, and even more, but the stools have nothing characteristic. We rarely see a slight admixture of pus or blood in them. Tubercle bacilli have been repeatedly discovered in the dejections, but the search for them is rather difficult. We must call attention, however, to the fact that many patients have diarrhea during life in whom we find at the autopsy no intestinal tuberculosis, but only a simple intestinal catarrh. Severe diarrhea of a persistent and refractory character may also occur in association with amyloid degeneration of the intestine, which is not infrequently seen in connection with other amyloid changes. Sometimes tubercular ulcerations of the intestine are found at autopsy which, during life, had caused no diarrhea.

In cases of severe intestinal tuberculosis we sometimes meet with meteorism. With deep ulcers, extending to the peritoneum, we often find marked tenderness of the abdomen.

The peritoneum may be affected by the tubercular ulcers of the intestine in a twofold manner. Genuine peritonitis from perforation, with a purulent or even a sanious exudation, is quite rare, and is excited by the rupture of an ulcer and the entrance of the contents of the intestine into the abdominal cavity. An infection of the peritoneum with the tuberculous poison is more frequent. This may arise from deep-seated ulcers, which do not reach actual perforation, so that we have a peritoneal tuberculosis or a tuberculous peritonitis. During life peritonitis from perforation and that from tuberculosis are not always to be distinguished. We must also mention that simple ascitic fluid is sometimes found in the abdominal cavity in phthisis, which may lead to a false diagnosis of peritoneal tuberculosis.

Another way in which we have a peritoneal tuberculosis in the course of phthisis is from the extension of the process in a tuberculous pleurisy, through the diaphragm to the peritoneum.

4. *Liver and Spleen.*—We very often find a few or even many tubercles in the liver in phthisis, but they have no clinical significance. The liver is almost always infected with the tuberculous poison from tubercular ulcers in the intestines, from which the poison passes to the branches of the portal vein and then to the liver. Fatty liver and amyloid or lardaceous liver are more important clinical changes. We can sometimes recognize the former by making out on physical examination the increase in the size of the organ, and by feeling its characteristic blunt lower edge. Moreover, we must emphasize the fact that, in our experience, fatty infiltration of the liver is found much more rarely at the autopsy of consumptive subjects than the statements of many earlier authors would lead one to expect. Still, the occasional finding of a pronounced fatty liver in an otherwise extremely emaciated phthisical body is a very striking though not yet fully explained circumstance. It would almost appear as if the fat had migrated from the tissues to the liver, and remained there in part in a nonoxidized state.

Amyloid degeneration of the liver appears almost always in association with amyloid change in other organs (kidneys, spleen, intestine). If the amyloid degeneration is advanced, the liver is considerably enlarged, and it is usually possible to feel distinctly its sharp and resistant edge; and, not infrequently also, its firm upper surface.

Miliary tubercles or single large tubercular nodules in the spleen have a pathological interest only. Great splenic enlargement is found in amyloid degeneration.

5. *Kidneys, Urinary Passages, and Sexual Organs.*—The presence of miliary tubercles in the kidneys is the first change in them to be mentioned, but it has no clinical significance. Extensive tuberculosis of the genito-urinary apparatus, however, may produce marked symptoms, such as pyuria. Genito-urinary tuberculosis will be discussed in a later part of this work. In regard to the symptoms of amyloid kidney, which may develop in the course of phthisis in connection with amyloid disease in other organs, we will refer to the section on renal diseases.

Genuine cases of nephritis, both acute and chronic, are also found quite frequently in phthisis, often combined with amyloid disease. We have also observed chronic, hemorrhagic nephritis. The extent to which the kidneys are affected cannot escape notice if the urine is carefully examined. The development of nephritis is probably always referable to the excretion of toxic material, arising from the disease in the lung.

6. *Circulatory Organs—Blood.*—The rate of the pulse is often increased in consumption. This increase in frequency may be merely proportional to the fever, if any exists. It is also usually seen and it may be considerable when there is no fever. A persistently rapid pulse, when the temperature is little, if at all, elevated, may therefore be an important diagnostic sign of pulmonary (and all other) tuberculous diseases. The increase of the pulse, which readily comes on from comparatively trifling external causes, is especially noteworthy. It may be seen after slight physical exertion, or upon mental excitement, as during the physician's visit.

Anatomical changes in the heart are rare, except that it is often remarkably small and flaccid. Moderate fatty degeneration of the heart, slight endocarditis of the valves, or occasional tubercles in the heart, cause no symp-

toms. The occurrence of tuberculous pericarditis, however, is important. This almost always arises from the extension of the tubercular process from the adjacent pleura, but in exceptional cases pericarditis has been seen as a result of rupture of a tuberculous lymph-gland or a pulmonary cavity into the pericardium. In many cases of tuberculosis the blood shows a simple anæmia. As a rule, the number of red blood cells is not lowered as much as is the percentage of hemoglobin. A moderate polynuclear leucocytosis, with a lowered proportion of lymphocytes, is often found in advanced tuberculosis with fever. In cases with severe, persistent dyspnœa (compensatory?) increase in the red blood cells and hemoglobin is sometimes observed.

7. *Lymph-glands.*—The lymph-glands are a favorite seat for tuberculous changes. We have stated above that the so-called scrofulous, cheesy lymph-glands, which are seen chiefly in the neck and the axillæ, are affected with tubercle in the majority of cases. The tuberculous infection probably develops here from slight injuries and excoriations of the skin, by which the bacilli enter the body and reach the neighboring glands by means of the lymph current. In other cases the infection comes perhaps from the mucous membrane of the pharynx. In tuberculosis of internal organs, too, we very often find the corresponding lymph-glands enlarged and more or less cheesy. The bronchial lymph-glands are swollen as a result of pulmonary tuberculosis, the mesenteric and retroperitoneal glands as a result of intestinal tuberculosis. The tuberculosis of the bronchial lymph-glands is of especial importance in children. Indeed, the tuberculous virus which has gained access to the lungs may apparently reach the bronchial glands by means of the lymph-channels, passing through without affecting the lungs themselves, and occasion a tuberculous disease of the glands. Bronchial glands, thus diseased, may involve the lungs by direct or indirect (via the lymph-channels) infection. Recently, as above mentioned (page 277), there has been a tendency to refer primary tuberculous disease, even in adults (in some cases, at least), to the bronchial lymph-nodes.

Pressure from the enlarged glands may affect the air-passages, the branches of the pulmonary artery, the veins, the recurrent nerve (paralysis of the vocal cords), and even the aorta. Perforation of the cheesy bronchial glands into the œsophagus, the blood vessels, etc., has also been observed. Tuberculosis of the bronchial glands does not present any definite type of disease, however. Very doubtful, too, is the interpretation of any statements concerning painful points in the back, or certain painful sensations felt in consequence of pressure upon the bronchial lymph-glands by means of a bougie in the œsophagus. X-ray examination (see Plate II) gives by far the most certain clew to the diagnosis of tuberculosis of the bronchial glands, if the plates are examined without bias by experienced men. The author himself observed a noteworthy case of tuberculosis of the bronchial glands, with compression of one vagus nerve in a patient who, during life, for weeks coughed up large amounts of a purely seromucous expectoration containing no bacilli.

8. *Nervous System.*—We have already mentioned various nervous symptoms in the description of the general symptoms. We must also add that tuberculous meningitis is seen at the termination of phthisis (see Vol. II), and that large solitary tubercles may occasionally develop in the central nervous system.

9. *Skin.*—We have spoken of the great tendency which many patients have to severe sweats, especially at night. The frequent appearance of pityriasis versicolor, especially on the skin over the thorax, is also worthy of note. We often see moderate œdema of the legs and ankles, which is due to weakness of the heart. More marked œdema of one leg sometimes arises from thrombosis of the femoral vein. We must also mention here, in conclusion, the specific tuberculous disease of the skin, lupus, and, in addition, the so-called cutaneous tuberculides. The manner of infection is very evident in some cases. Thus, for example, in a woman who had for a long time washed the soiled handkerchiefs of a very sick consumptive, I observed a tuberculous skin lesion of the nature of the so-called "post-mortem tubercle," formerly so commonly seen among pathologists. In this case the source of infection was very evidently the sputum. The details of the different types of tuberculous skin disease are to be found in special works on the subject.

DIAGNOSIS

The diagnosis of pulmonary tuberculosis has become absolutely certain, since the discovery of the tubercle bacilli, by the demonstration of their presence in the sputum (*vide supra*). In all incipient cases, in which the other symptoms of the disease have not yet made themselves manifest, but the suspicion of incipient phthisis has been aroused by a persistent cough, by marked pallor and emaciation, by slight hoarseness, by an evening rise of temperature, by the appearance of night-sweats, by the presence of a hereditary predisposition, and similar symptoms, the finding of tubercle bacilli in the sputum is often the sole deciding factor. If no sputum can be obtained, the exhibition of sodium iodid may stimulate expectoration. Still, it must not be forgotten that the examination for bacilli is decisive only when its result is positive, and that the greatest attention should be given also to all the other symptoms. To form an opinion as to the severity of a particular case, and as to its exact distribution and the variety of the tubercular process, is at present possible only by means of a consideration of the other symptoms, and in particular of the results of physical examination. The latter, therefore, has lost none of its importance by the discovery of the tubercle bacilli. Confusion between phthisis and other diseases is twofold. Where the constitutional symptoms are predominant, and there are no marked pulmonary symptoms, an existing tuberculosis may be overlooked. In the beginning, especially, many cases of phthisis are considered to be merely anæmia, chronic gastric catarrh, or simple bronchitis. If a continuous or intermitting fever appears in an early stage of phthisis, before any marked pulmonary symptoms have developed, the disease may be mistaken for malaria or the like. On the other hand, it is by no means rare to consider patients phthisical who are suffering from some entirely different affection. Severe latent diseases of the stomach, or certain general diseases, such as anæmia, diabetes, or chronic nephritis, may be mistaken for phthisis. Other pulmonary affections, too, may be confounded with tuberculosis, especially chronic bronchitis, emphysema, bronchiectasis, fetid and gangrenous processes, and carcinoma of the lungs. A careful, unprejudiced, and complete examination of the patient is the only possible protection against such errors. It is very important for the

physician to know that there is such a thing as hypochondriacal phthiseophobia. Some nervous individuals are tormented by a constant dread of becoming consumptive. Such persons complain of thoracic pain, dry cough, weakness, and other imaginary symptoms, which might readily mislead the physician into supposing that they actually have incipient pulmonary tuberculosis. A mistake is especially apt to be made if the patient claims to have suffered from hemoptysis. The experienced physician will usually be struck by the fact that the "hemoptysis" is said to have recurred in slight amounts almost daily, and has lasted for weeks. A thorough examination usually discloses the nasopharynx or the gums as the source of the bleeding (compare with remarks on the so-called hysterical hemoptysis in Vol. II of this work). Of course, the correct interpretation of such conditions is usually no difficult matter to the experienced physician, who is guided by the general impression of "nervousness," and by the complete absence of any objective physical signs.

In this connection we ought, furthermore, to express our opinion of the diagnostic value of Koch's tuberculin (*vide infra*). Koch made the important discovery that tuberculous patients, particularly those suffering with pulmonary tuberculosis, exhibit a particular "reaction" after the injection of small amounts of tuberculin (gm. 0.001 to 0.002 of Koch's preparation). When this reaction occurs there develops, some four or five hours after the injection, a fever of 102° to 104° F. (39° to 40° C.), associated with chilliness, headache, pain in the limbs, nausea, and languor. This constitutional reaction passes off after some twelve or fifteen hours. If the tubercular process is located in the skin, trachea, or some other part where it is accessible to direct observation, a very decided local reaction may usually be observed besides the constitutional disturbance. The tuberculous tissue becomes swollen, red, and finally in part necrotic. In patients with tuberculosis of the internal organs, also, this local reaction probably takes place, but of course it cannot be actually seen, although it may, perhaps, be inferred because of the secondary phenomena. Thus, in pulmonary tuberculosis there may be an increase in the cough and expectoration. On the other hand, if an injection of tuberculin is made in a person who is healthy, or who is suffering from some disease other than tuberculosis, then, according to Koch, there will be no reaction at all to small doses. To obtain any reaction in persons who are healthy, or, at any rate, not tuberculous, the dose of Koch's preparation must be increased to the amount of gm. 0.01.

These statements of Koch are, in general, certainly true, but subject to several exceptions. Other observers as well as the present author have occasionally found that healthy persons, or persons without any demonstrable focus of tuberculosis, have reacted to doses of 1 or 2 mgm. of tuberculin; while, on the other hand, patients with indubitable pulmonary tuberculosis have shown no distinct reaction even to considerable doses of tuberculin. Consequently, the result of an injection of tuberculin can never be completely decisive from a diagnostic point of view, and in practice, therefore, the employment of tuberculin for diagnosis has been adopted by comparatively few.[1] Still, it would be a mistake to discard this means of diagnosis completely. In doubt-

[1] In veterinary practice, the tuberculin test is employed with very excellent results in the diagnosis of bovine tuberculosis.

ful cases the injection of from 1 to 2 mgm. of tuberculin is of real importance, and the result of the test is certainly a valuable item in summing up such a case. It is presupposed that the patient's temperature is carefully taken for two to three days (and every two hours, if possible) before the tuberculin injection is given. If the temperature has been entirely normal, the injection of even a small dose of tuberculin (0.5 mgm. or less) will be followed by a distinct, if slight (1° to 2° F. [0.5° to 1° C.]), elevation of temperature. But it is just the fact that the tuberculin test can only be employed in patients free from fever that diminishes its practical importance. For often it is precisely in patients suffering from fever that we wish to learn if there is a hidden focus of tuberculosis.

Very interesting, too, are the observations recently made, in which the inoculation of tuberculin was also found to have a local effect differing according to whether the individual is suffering from tuberculosis or not. According to Pirquet, if a drop of a twenty-five-per-cent, or even weaker, solution of tuberculin be introduced into the skin through a pin-point hole or a superficial scratch, when tuberculosis is present in an individual, a distinct cutaneous reaction, consisting of more or less hyperæmia and swelling (see Fig. 55), will appear within twenty-four to forty-eight hours at the site of the vaccination. In the absence of tuberculosis there is no such reaction whatever. Calmette and Wolf-Eisner perform the test by dropping a single drop of a one-per-cent solution of tuberculin into the conjunctival sac of the eye. An ofttimes rather intense inflammatory redness of the conjunctiva (ophthalmic reaction) appears after twenty-four hours in cases of tuberculosis, and is absent in healthy individuals. This reaction has not been widely employed in medical practice, because very disagreeable inflammations

FIG. 55.—Pirquet skin reaction (personal observation). *a*, Reaction at the site of the tuberculin inoculation. *b*, Control scratch without tuberculin inoculation.

of the eye have occasionally been observed after its use. Its diagnostic value is also limited, because it is occasionally absent in undoubted cases of tuberculosis and present in individuals in whom tuberculosis is not even suspected. Although the practical diagnostic value of both the above-mentioned tests cannot, as yet, be rated very highly, they are of great theoretical interest, and certainly deserve thorough further study.

Finally, we would also refer to the great diagnostic value of X-ray examination (see Plate II) in pulmonary tuberculosis, especially in the estimation of doubtful and of incipient cases. When careful technic is employed, tubercular infiltrations of the lungs give very distinct shadows, but a trained eye is certainly just as essential for their interpretation as is a trained ear for the proper detection of differences in the percussion note. Numerous observations in recent years have proved to me that all tubercular infiltrations are easily recognized in the X-ray pictures, and usually correspond exactly with the changes in percussion; occasionally, however, they may even stand out prominently where percussion has revealed only doubtful changes or even none at all. X-ray examination is, at any rate, an extremely valuable procedure in the establishment of a diagnosis. The fact is also very important that tuberculous bronchial lymph-nodes that can be demonstrated in no other way often appear very distinctly in X-ray pictures (P. Krause and others). Their presence should put us on our guard, even though the lungs are otherwise normal. On the other hand, when the possibility of incipient tuberculosis is being considered, there is nothing that gives one such ease of mind with respect to the normal state of the lungs as the demonstration of a completely uniform clearness in the X-ray picture. Naturally, it must be conceded that under some circumstances beginning tuberculous changes in the apices (so-called "apical catarrh") can be demonstrated by auscultation when it does not cast any distinct X-ray shadow. The so-called Williams's sign (early limitation or absence of diaphragmatic movements on the diseased side) is not of great importance, inasmuch as it is but rarely observed. When not due to pleuritic changes, it appears to be due to a lesion of the phrenic nerve at the apex of the lung.

At the conclusion of these remarks on diagnosis, I would point out that the diagnosis of incipient tuberculosis should never be based on a single symptom, but on the study of the whole case. After several days' careful observation of the temperature, the pulse, physical signs, sputum, X-ray picture, and perhaps the tuberculin reaction, a correct opinion can usually be arrived at.

PROGNOSIS

It is very difficult to make a general statement as to the prognosis of pulmonary tuberculosis. There is no doubt that tuberculous foci in the lungs, if of limited extent, may become completely healed, at least from a clinical point of view. Indeed, such recovery probably takes place oftener than many suppose; at least, it is not very exceptional to find at the autopsy of elderly persons cicatricial contractions in the apices of the lungs which may, without doubt, be regarded as healed tuberculosis. In many of these cases the previous tuberculosis had never come to the knowledge of any physician. At the present day the discovery of tubercle bacilli in the sputum has rendered the

FIG. 1.—RADIOGRAM OF AN ADVANCED CASE OF PULMONARY TUBERCULOSIS. Extensive involvement of the left lung and, to a lesser degree, of the right.

FIG. 2.—RADIOGRAM OF A CASE OF MARKED TUBERCULOUS DISEASE OF THE LEFT LUNG. Large cavity at *c*. Slighter involvement of the right lung.

FIG. 3.—RADIOGRAM OF A CASE OF BEGINNING PULMONARY TUBERCULOSIS. Both sides show distinct shadows in the neighborhood of the bronchial glands.

diagnosis of even very limited tubercular processes an easy matter, and, consequently, cases of recovery from pulmonary tuberculosis are now recognized much more often than formerly.

Nevertheless, the prognosis of pulmonary tuberculosis must always be regarded even now as very serious, and when the disease has made any great progress the prognosis is decidedly unfavorable. Many cases of apparent recovery exhibit merely a temporary improvement, to grow worse again, and the assertion that treatment is absolutely hopeless in almost all cases in which the disease has reached an advanced stage is, unfortunately, too well established to require elaborate argument.

There is, however, one fact of extreme importance which should never be disregarded in the prognosis of pulmonary tuberculosis. There is a great difference in the duration of the disease. It is possible for the process to go on for years, while the patient feels tolerably well, and yet the disease smolders. From this point of view cases may be distinguished as "benign," compared with those of rapid progress, and this prognostic distinction has the greatest importance in practice, although it is often very difficult to recognize. Many a consumptive, when first examined, gives one the impression that he cannot live a fortnight longer, and yet the disease lasts many months, with improvement in most of the symptoms and general alleviation of the patient's condition. Again, one believes the disease to be incipient, encourages the family, and the patient dies in a few weeks of galloping consumption.

Certain complications may occur which it is impossible to foresee, such as pulmonary hemorrhage, pneumothorax, tuberculous meningitis, and miliary tuberculosis. Apart from these, however, there are certain conditions which lead us to expect a comparatively favorable course for the disease: such are a vigorous constitution unimpaired by bad habits, of which drinking is particularly unfavorable; a good weight; freedom from hereditary taint; limited extent of the local process, and moderate age [experience has shown that all cases of tuberculosis beginning in individuals, say, over thirty, give a better prognosis, in general, than do those beginning in earlier life]; the absence of complications; and, finally, the persistent absence of fever. This last factor is so important that we must lay special stress upon it. If there is no fever whatever in a case of pulmonary tuberculosis, the patient is at that time in a condition in which the disease is quiescent; and a decided improvement, perhaps even cure, may be expected if appropriate means are employed. On the other hand, whenever fever appears we know that the disease is not quiescent, but is actively advancing with more or less rapidity. The important points about the temperature in this regard have been already emphasized (see page 298). It is self-evident, also, that the worldly circumstances of the patient, as well as the factors which we have just alluded to, are of great prognostic importance. On these, for instance, depends the possibility of proper care, of abundant nourishment, and, it may be, of a change of climate.

TREATMENT

1. **Prophylaxis.**—The question of what prophylactic measures may effectually prevent the extension of the disease has entered upon a new stage since our definite knowledge as to the infectious nature of tuberculosis. We can

no longer doubt the contagious character of phthisis, in support of which isolated examples were previously brought forward. Even if, according to all experience, the danger of contagion is not very great, still, it is foolish to ignore it entirely. We must therefore call the attention of the relatives of phthisical patients to the possibility of this danger, and we should not permit the children of such patients to be uselessly exposed to it. We should take satisfactory precautions for isolation, and also for disinfection of the sputum. Suitable sputum cups should be employed, and care should also be taken to prevent the dissemination of the expectoration, either in its fresh state or after it has become dry. The future will teach us whether much evil may not be averted by such measures.

The "prophylaxis" till now employed was almost exclusively confined to hardening and strengthening the threatened individual as much as possible. We should try to strengthen the bodies of children of a weak habit, with "scrofulous" symptoms, and children from families in which cases of tuberculosis have already occurred, and thus to arm them against the enemy that threatens them. Good food, fresh air, and a diminution of the sensitiveness of the body by cold sponging and cold baths—these are factors whose favorable influence is generally recognized, but should also not be overestimated.

The removal of certain foci of tuberculous disease, already existing, from the body may prove of great prophylactic importance. We refer to the timely treatment or extirpation of scrofulous—that is, tuberculous—swellings of the lymph-glands, healing or resection of tuberculous bones and joints, etc. Although in individual cases we can, of course, never know whether the part removed is the sole focus of disease in the body, still, we are undoubtedly justified in trying to remove at least one possible source of some later general infection. A fuller discussion of this important point must be left to the works on surgery.

2. **Therapeusis.**—Physicians have often thought that they had discovered a specific remedy for tuberculosis, but apparently they have thus far been mistaken. Formerly, the inhalation of antiseptic substances, such as carbolic acid, benzoate of sodium, lignosulphite, and iodoform, was recommended, but this practice is now almost entirely given up. Arsenic was for a time much used, best given in pills containing $\frac{1}{20}$ gr. (gm. 0.003) repeated several times a day; but this practice, again, has not held its own. Arsenic may be tried in incipient cases, particularly those associated with marked anæmia, but great curative influence is not to be expected from it. Creosote has won far more advocates. Continued for a considerable time in large doses (15 to 30 gr. [gm. 1 to 2] or more, in the course of twenty-four hours), it is regarded by many physicians as an excellent remedy in incipient and even in advanced pulmonary consumption. It is best prescribed in gelatin capsules, or in a mixture of 1 part of creosote to 2 parts of the tincture of gentian, of which 20 to 80 drops may be given three times a day in a considerable amount of milk, or in wine. This remedy is usually fairly well borne, and the patients are pleased with the improvement it causes in their appetite and general condition. Of late, guaiacol, the active ingredient of creosote, has been generally used instead of the drug itself, especially since carbonate of guaiacol has been brought forward, a preparation which has very little of the disagreeable odor and taste of creosote itself. Carbonate of guaiacol is administered

in powders containing about 8 gr. (gm. 0.5), in daily doses at first of 25 gr. (gm. 1.5), gradually increasing to 30 or 45 gr. (gm. 2 to 3). Dyspeptic disturbance sometimes follows the use even of guaiacol, but still this remedy is, in general, better borne and more readily taken than creosote. Thiocol (potassium sulphoguaiacolate) is another creosote preparation worthy of trial. It is taken in the form of powders, gr. vijss. to xv (gm. 0.5 to 1.0), several times a day, or in the form of a proprietary solution known as sirolin. The following mixture is often employed in my clinic: Potassium sulphoguaiacolate, 20 parts; syrupus aurantii, 20 parts; distilled water, 200 parts. One half to one tablespoonful of this is to be taken three times a day. As to the specific and therapeutic use of creosote and its numerous derivatives (in addition to those above mentioned, creosotal, sulphosote, eosote, and many others), in pulmonary tuberculosis, it is not easy to form a decided opinion. It is certain that many patients praise these remedies, and improve considerably while using them; but, on the other hand, the indiscriminate laudation of many authors is decidedly unjustifiable. When the disease is slowly progressing with persistent subfebrile temperatures, the author has scarcely ever felt sure of any distinct influence exerted by guaiacol or creosote upon the temperature and the disease. Nevertheless, it is often advisable in practice to make trial of these remedies, particularly of the carbonate of guaiacol. The remedy must be employed for months and, if possible, in increasing doses. Among the remedies which are recommended as having a specific influence, we will also mention cinnamic acid, which is contained in Peruvian balsam. This has been employed extensively by Landerer in the treatment of various tuberculous diseases, and particularly in pulmonary tuberculosis. He gives it in the form of small intravenous injections of a five-per-cent emulsion, made up of the sweet oil of almonds and the yolk of egg. The results obtained by Landerer, in the institute of Krähenbad, in the Black Forest, appear very favorable, but as yet they lack confirmation. The method has not yet been generally adopted.

On the basis of our present general bacteriological knowledge, an entirely different scientific significance belongs to the ever-increasing efforts of recent years to discover a true, specific curative agent for tuberculosis. At the end of 1890 great interest was excited, as is well known, by the statement of R. Koch that he had extracted from pure cultures of tubercle bacilli, by means of glycerin, a substance called tuberculin, by which he was able to heal tubercular processes both in animals and men. This assertion aroused the thoughtless enthusiasm which is unfortunately so common with regard to therapeutic questions, and which indeed in this case was rendered excusable by the high authority of the discoverer. In fact, within a few weeks after the remedy was made known, numerous reports were published of cures due to tuberculin. But the longer the experiments were pursued the more evident it became that these extravagant opinions were not substantiated, and there set in a revulsion of sentiment which has led to many adverse criticisms, perhaps equally unjustifiable with the early praise. It is absolutely impossible to speak of tuberculin as an established remedy for tuberculosis. In many patients there does indeed appear a decided improvement under treatment, but these cases are such as were favorable anyway, and therefore they may owe their improvement to the general symptomatic treatment and regimen

which they have also enjoyed. At any rate, tuberculin has done no harm in such cases. Then, again, we have seen numerous cases of rather severe disease, in which it was not possible to notice any distinct influence of the tuberculin upon the general course of the illness. Perhaps the condition of the patient improved or remained unchanged; or, again, it grew worse. Yet, whatever the change, it was such as might have occurred independently. Finally, there are, in the third place, cases which have been seen by the author as well as other observers, in which there was such a decided change for the worse shortly after the commencement of treatment, that one might really suppose that the remedy had exerted an actually harmful influence. We refer especially to cases in which the patient, previously without fever, exhibited after the injection a persistent elevation of temperature, with a more rapid advance of the local process in the lungs.

Consequently, we may say that, in all advanced cases, we can expect nothing from tuberculin. In incipient cases we may make trial of it, but we should be extremely careful in our dosage, so as to avoid any chance of doing injury. Koch's rule at first was that the physician should begin with injection of gm. 0.001, and gradually increase. In this way was discovered the interesting fact that the necessary dose for the production of the "reaction" (*vide supra,* page 306) became greater and greater. Many patients would finally bear the injection of 100 mgm. of tuberculin without any reaction at all. At first it was regarded as the object of treatment to reach this point of tolerance, but the practice is now abandoned. One should begin with very small doses, about 0.25 to 0.5 mgm., and increase so slowly and by such minute gradations as to avoid any marked reaction. By such a method we can almost entirely exclude any harmful effect, and it may not be impossible that some of the patients who have had a favorable result actually owe their improvement to the tuberculin, but it is extremely difficult to make sure of this. It would require the continuous observation of many patients for years to enable one to form a reliable opinion with regard to this question. Still, it cannot be denied that more and more observers have recently spoken in favor of tuberculin. In addition to "old tuberculin," various other tuberculin preparations (new tuberculin, Spengler's bovine tuberculin, etc.) and curative sera have been presented. We are, however, far from any conclusion as to their value. Yet, when everything is considered, including other bacteriological facts, we seem to be standing on the threshold of a future in which the remarkable effects of bacterial products will be made to exert a curative influence on the infectious diseases in general, and, especially, on tuberculosis.

Meanwhile, therefore, we physicians must continue to lay the greatest stress in the treatment of tuberculosis upon those methods which are termed in the widest sense hygienic and constitutional. It is undeniable that, by the correct and faithful employment of such methods, many very satisfactory successes may be obtained.

The hygienic and constitutional method of treating consumption has for its object the greatest possible promotion of the natural powers of healing. We aim at this, in the first place, by avoiding as much as possible all influences which might cause a further extension of the disease, and by reënforcing, so far as we can, all influences which increase the resisting powers of the individual, and the processes of spontaneous cure. The factors which

are of most importance in this connection are, first, diet; second, rest; and third, the uninterrupted enjoyment of good air. To obtain these three therapeutic factors all at once requires the renunciation by the patient of his calling, and his usual mode of life. The treatment of tuberculosis, therefore, should begin with the demand that the patient, for as long a time as possible, should devote himself exclusively to the care of his health. The next point is the choice of the place in which the "cure" shall be carried on. In many cases the proper management of the patient may be pursued under the ordinary surroundings of home. Often, however, this is not the case, because the last two, or even all three, of the essentials named cannot be provided at home. It, therefore, is the duty of the physician in every individual case to determine how the desiderata are best to be obtained. According to the patient's means we consider respectively a residence in the country (if possible, in a picturesque and wooded region); a special health resort; or, finally, a suitable sanitarium. From a therapeutic standpoint, there is no doubt that treatment in a specially adapted sanitarium is chiefly to be recommended. In this all the requirements for recovery can best be carried out, and the patient constantly remain under medical observation. In most cases it is only external considerations, for instance, pecuniary, which keep the patient from a sanitarium. It is a true philanthropy, therefore, which has of late led everywhere to efforts to render the benefits of institutional treatment accessible to the less wealthy classes. The requirements for proper treatment are also partially fulfilled in the so-called public health resorts for pulmonary consumptives. These resorts, however, have the disadvantage that the patient is left far more to his own resources, and is consequently apt to be incautious, and hence jeopardize his recovery. A public health resort would, therefore, be especially chosen for patients who have already been in an institution, and have learned what mode of life is proper for them; or for those who have already so far recovered that they may be allowed a certain degree of freedom.

With regard to the minutiæ in carrying out the above-enumerated essentials of treatment, we would add as follows: 1. *Diet.* This should be as nourishing and abundant as possible. Meat, milk, eggs, farinaceous foods, and butter are chiefly to be recommended, care being taken that the body should receive a sufficient amount of carbohydrates and fats, as well as an abundance of albumen. Many special "cures" for pulmonary consumption have a value, in so far as they lead to the ingestion of an abundant amount of easily assimilated nourishment. Such are cures with milk, koumiss, or kefir. It has even been proposed to introduce large amounts of such nourishment as milk and pulverized meat into the stomach by means of a stomach tube, and thus accomplish "overfeeding." This method has not become very popular, although it may be indicated in some cases. It is very important to see that the patient has a diet that is not only abundant, but also palatable and varied. If pure milk is not readily taken, we may try the addition of coffee, tea, common salt, or brandy. With regard to the prescription of alcohol we recommend, unhesitatingly, moderate amounts of beer, and particularly such beer as is rich in extractive matter; and perhaps also extract of malt, and porter. Small amounts of good wine may contribute to the improvement of the appetite and the general condition. On the other hand,

we think it useless and, in some circumstances, harmful, to order large amounts of the stronger alcoholic beverages, such as port wine and brandy, as prescribed in many sanitariums. Artificial foods, such as various preparations of meat, somatose, neutrose, hygiama, and the like, should be used merely as makeshifts. The employment of cod-liver oil to the amount of 2 to 4 tablespoonfuls a day is sometimes not inappropriate, if it is well borne. 2. The two other factors of cure—*rest, and the continuous enjoyment of good air*—are best fulfilled by the fresh-air treatment, which is of late gaining in importance and popularity. The patients spend the greatest part of the day lying in the open air on comfortable reclining chairs. At the same time they avoid any unnecessary bodily exertion, any great demands upon respiration, and any irritation of the respiratory passages. Gain in bodily weight is promoted. The limitation of the diseased process in the lungs is favored. Of course, in such matters also the individual should be considered, for moderate exercise in the open air is certainly for many patients not harmful, but rather beneficial. This applies only to phthisical patients who have no fever. As soon as there is even the slightest elevation of temperature, complete rest is the only correct procedure. Often the methodical carrying out of the open-air treatment under suitable conditions demands the resources of a sanitarium, but we may find in a garden or on a veranda a sunny spot, screened from the wind, where the patient may lie comfortably and pass the entire day till near sunset in the open air. In case of necessity the patient must content himself with a place by an open window. The advantages of the climatic health resorts (*vide infra*) consist principally in the fact that they render possible a continuous enjoyment of the open air, even during the colder part of the year. It has been maintained that climatic factors, particularly elevation, exercise a specific influence upon the healing of pulmonary tuberculosis; but this has not yet been proved.

The best-known sanatoria for pulmonary diseases are Görbersdorf in Silesia, St. Blasien, Wehrawald, and Schömberg in the Black Forest, Reiboldsgrün in Hohenhonnef on the Rhine, Andreasberg in the Hartz Mountains, Davos and Arosa in Switzerland, Gardone, Gries, etc. Of the public health resorts which are particularly suitable for consumptives, we would name for summer use the acidulated, alkaline, and chlorid-of-sodium waters of Ems, Obersalzbrun, and Reinerz; the chlorid-of-sodium waters in Reichenhall, Salzungen, and Soden; the mud springs in Lippspringe, Inselbad, and Weissenburg in Switzerland. For summer climate, the following are to be recommended: Beatenberg, Heiden, Engelberg in Switzerland; Badenweiler, St. Blasien, Rippoldsau in the Black Forest, and many others. For the colder portion of the year we have, in their lofty situation, Davos, Arosa, and others. The more vigorous the constitution of the patient, the more proper it is to recommend him to go to a great elevation in the winter; while delicate subjects are usually better suited in the southern resorts. Of course only the very distant health resorts in Algiers, Egypt, Malta, and the much-praised Madeira, can furnish a certain guarantee of constant mild weather. The Sicilian health resorts (Catania and Palermo), and also Ajaccio, afford favorable climatic conditions; while the health resorts of the Riviera, Meran, Gries, Arco, Gardone, Lugano, Pallanza, and Montreux, are much more uncertain in this respect, and therefore are to be used merely as stopping places by

the way during the spring and autumn months. We would also here state that, in incipient cases that have become stationary, a residence by the sea, or a long sea voyage, may sometimes be of great help.

We cannot go into a full description here of all the health resorts mentioned. We cannot omit, however, calling special attention to the fact that we should always ask ourselves, in choosing a health resort, whether the expense and inconvenience thus imposed upon the patient can be balanced by the possible benefit. The earlier the stage of the disease and the better the general condition of the patient, the more will his physician be justified in urging him to make every sacrifice in order to regain his health. We must particularly insist with the patient that no cure of tuberculosis can be accomplished by a single visit to any health resort, but that recovery must be achieved by living continuously for years in accordance with all the demands of hygiene. On the other hand, it is wrong, both from a medical and from a humane standpoint, to send away consumptives in the last stages of their disease, to perish far from home and friends. Consumptives with fever should never be sent from home, unless they find refuge in a real sanitarium where they may enjoy the constant observation and treatment of a physician.

[Our own health resorts for consumptives are too well known to demand extensive consideration here. The prime object is to secure for the patient a pure air, with such climatic conditions that he can pass the largest amount of time out of doors, at the same time that within doors his comfort is provided for, and a sufficiency of suitable and well-cooked foods is attainable. In Colorado, New Mexico, and California large numbers of former consumptives are leading active lives. Florida, Aiken, Thomasville, Asheville, and some other southern resorts, are good winter asylums for many cases, but patients should not return to New England before June 1st. An outdoor life in the Maine or Adirondack woods during the warmer months is highly to be recommended for early and otherwise suitable cases. Saranac, N. Y., affords every comfort during the colder months, combined with the very best medical attendance, and many people do well there. A relative disadvantage under which nearly all American health resorts labor, as compared with those of Europe, consists in the greater difficulty in providing occupation and thus securing a mental attitude most favorable to recovery. In general, the northern seaboard is much less favorable than the interior, and early cases often do well during the winter removed from the dampness of the coast, with its alternations of freezing and thawing. A change of climate is a very important step, and should receive the most careful consideration of the physician—the circumstances of the patient, the stage and character of his disease, his tastes, etc., being carefully weighed before a decision is reached.]

Finally, the employment of hydrotherapy should be mentioned in the hygienic treatment of tuberculosis. Although this cannot exert any specific influence on the disease, and therefore must not be prized too highly, yet it is often beneficial in the form of cold sponging or brief cool douching and the like; and the stimulation of the skin has a favorable influence upon the general health. In severe febrile cases we may order sponging and rubbing in bed with cold water, brandy, aromatic vinegar, and, in addition, chest packs, compresses, and the like. Also for the relief of symptoms (*vide infra*), such as pain, fever, and perspiration, we may often employ sponging, cold or warm

compresses, and the wet pack ("Priessnitz"). I not infrequently employ inunctions of soft soap for tuberculosis, similar to those used for a long time in the treatment of tuberculous pleurisy and of swollen lymph-glands.

The symptomatic treatment of phthisis is directed in the first place against the pulmonary symptoms. We use much the same remedies to help the cough as in chronic bronchitis. We try inhalations [1] with a solution of common salt, or of the alkaline carbonates, or, if there is much secretion, with solutions of tannin and the balsams, such as turpentine, or balsam of Peru. When there is severe, spasmodic cough, inhalations with narcotic solutions sometimes give some relief, such as cherry-laurel water, opium, or bromid of potassium.

Morphin stands first among the drugs employed to check the cough. We should be cautious and sparing in its use at first, but it is an indispensable remedy in severe and hopeless cases. It relieves the irritation of coughing, the pain and the oppression in the chest, and at least gives the patient for a time the desired sleep. In chronic cases, with moderately severe symptoms, we may use for a long time the milder narcotics with advantage, particularly codein (powders of gr. ss. to j [gm. 0.03 to 0.05], or phosphate of codein, gr. vijss. [gm. 0.5], aq. amyd. amar., 3v [20 c.c.], dose 15 to 20 drops); also the two morphin derivatives, heroin (gr. $\frac{1}{10}$ to $\frac{1}{5}$ [gm. 0.006 to 0.012] carefully given), and dionin (gr. 0.25 to 0.5 [gm. 0.01 to 0.03] a dose). Under some circumstances, we also employ the extract of hyoscyamus (for example, extracti hyoscyami, 1 part; aquæ laurocerasi, 20 parts; 15 to 20 drops of the mixture every two hours), the extract of belladonna [2] (in 0.5 to 1 gr. [gm. 0.03 to 0.05] powders), etc. It is important that the patient should learn to suppress his inclination to cough, at least up to a certain point. Sips of cold water often quiet the cough, as may also a "cough drop" held in the mouth, or a pastille containing the salts found in Ems mineral water.

If the patient complains of difficulty in loosening the expectoration, we prescribe expectorants, the action of which often fails to meet our desires, but which cannot be dispensed with in practice. The expectorants most frequently used are carbonate of ammonia, ipecacuanha, apomorphin, and senega. We very often combine expectorants with narcotics, as in Dover's powder.

If severe pain in the chest comes on, we often use local applications: mustard plasters, warm poultices and cold compresses, painting with iodin, or embrocations of chloroform. Narcotics, such as morphin, are indispensable in severe dyspnœa, which usually occurs only in the last stages of the disease or as a result of pneumothorax.

The treatment of hemoptysis is important. As a slight admixture of blood in the expectoration often precedes a severe hemopytsis, such an appearance always suggests caution. When there is any hemoptysis, absolute rest in bed is necessary. We should avoid any careful examination of the lungs, especially any severe percussion. We should lay a flat and not too heavy ice bag over the lung on the side from which we suspect the hemorrhage; the cold is usually well borne, but sometimes it aggravates the cough, and must then be omitted. We would also recommend swallowing bits of cracked

[1] For details as to dosage, see Appendix III, "Formulæ," in Vol. II of this work.

[2] Ext. belladon., U. S. P., is four times stronger than the German preparation.

ice. Narcotics, such as morphin, are the most suitable internal remedies, since they aid the cessation of the hemorrhage by suppressing the attacks of coughing. The more troublesome the cough the more necessary is it, therefore, to administer morphin in solution, by the mouth or even subcutaneously. Of remedies to check the blood we should name, first, extract of ergot or ergotin, which may be given by the mouth, or, still better, subcutaneously, in doses of 2 to 8 gr. (gm. 0.1 to 0.5) several times a day. Of the individual ergotin preparations, that to be most recommended, especially for subcutaneous use, is cornutin. citricum (Merck). [Any preparation of ergot will raise the tension in the pulmonary artery, and is therefore to be avoided. Inhalation of nitrite of amyl has recently been recommended. Its physiological action is, of course, the opposite. Purgation is advisable—e. g., an ounce of Epsom salts.] The subcutaneous injection of sterilized solutions of gelatin has been widely praised. Remarkable observations have recently been made on the hemostatic effect of serum injections. We may also try the fluid extract of hydrastis, with equal parts of an elixir, giving 30 to 40 drops of the mixture several times a day; also, acetate of lead and atropin. The influence of these remedies, particularly those last named, is, however, quite uncertain. The inhalation of a one- to two-per-cent solution of perchlorid of iron usually excites cough, and is therefore more harmful than beneficial. A very popular remedy among the laity, and one almost always at hand, is common salt, of which several teaspoonfuls may be given in water. When the hemorrhage recurs frequently, it is also advisable to "tie off" the limbs—that is, to apply bandages rather firmly around the middle of the upper arms and the thighs. This causes venous congestion, and hinders the return of blood to the lungs. When pulmonary hemorrhage has occurred, the diet should consist at first of nothing but cold milk, eggs, and similar food. Hot food, alcohol, and large amounts of meat are to be forbidden. It is a good thing to give acids, such as lemonade, or aromatic sulphuric acid, well diluted.

Even when the bleeding has ceased we must keep the patient several days in bed, and for a longer time be extremely cautious, since the hemorrhage is apt to be repeated.

The hectic fever of consumption is remarkably little influenced by antipyretic drugs. It often is entirely useless, or even, on account of its evil effect upon the stomach, actually harmful to give large doses of antipyretics to feverish consumptives, for long periods of time, particularly as the fever often of itself has deep morning remissions. The prescription of antipyretics is only justified when they make the patient feel better. The continuous use of pyramidon (gr. 4 [gm. 0.25]), three times a day, is particularly worthy of trial. Antipyrin, quinin, and other similar drugs may also be employed. On the other hand, it is very appropriate to give a cold sponging and rubbing to the whole body or chest, with water or alcohol, especially in the evening when the fever is high. The sponging is almost always well borne, and makes the patient feel brighter and easier. The cold pack may also be tried occasionally.

Cold sponging often diminishes the troublesome sweats in phthisis, but if this does not check them, we may often prescribe atropin to advantage, gr. $\frac{1}{120}$ to $\frac{1}{60}$ (gm. 0.0005 to 0.001) at night, but its action does not usually last very long. Lately agaricin in $\frac{1}{12}$- to $\frac{1}{6}$-gr. pills (gm. 0.005 to 0.01) has been

recommended for the night-sweats in phthisis; also picrotoxin, of which gr. $\frac{1}{8}$ to $\frac{1}{6}$ (gm. 0.008 to 0.01) is given in pill or solution at bedtime, and lately camphoric acid, gr. 20 to 30 (gm. 1.5 to 2) in wafers. Dusting the body with a powder of 5 parts of salicylic acid to 95 of French chalk is also good. Sage tea is a favorite remedy for night-sweats—2 or 3 cups of it cold at night—and so are milk and cognac.

If there is loss of appetite, small doses of quinin, compound tincture of cinchona, wine of cinchona, and other bitter remedies, such as tinctura amara (P. G.), are sometimes of service. It is also frequently a good thing to prescribe a little muriatic acid, 5 to 10 drops of the dilute acid, with the meals. It is often very hard to check diarrhea in phthisis. Opium, combined with tannin or acetate of lead, is most effective. This subject will be discussed more fully in the chapter on intestinal tuberculosis.

We often prescribe preparations of iron, combined sometimes with quinin or arsenic (*vide supra*), in the beginning of the disease to improve the general condition and the anæmia, but, as experience shows, iron is contraindicated in patients who are feverish or who have a tendency to hemoptysis.

The treatment of the diseases complicating phthisis is to be found in the appropriate chapters.

CHAPTER VII

ACUTE GENERAL MILIARY TUBERCULOSIS

Ætiology.—Acute miliary tuberculosis is a form of tuberculosis which we are justified in describing particularly because of its anatomical relations and its peculiar clinical history. The disease is characterized anatomically by the extremely abundant development of miliary tubercles in a comparatively short time in many organs of the body. We cannot liken this process to anything but an overwhelming of the body with tubercle bacilli, which in some way reach the different organs simultaneously, and in them give rise to the eruption of tubercles. Buhl advanced the hypothesis a long time ago, that a cheesy focus could be found somewhere in the body in every case of acute miliary tuberculosis, and that the general infection of the body resulted from the absorption of these cheesy masses by the blood. Later investigations, however, have given us a much more definite explanation of the nature and manner of this general infection. Ponfick found, in some cases of acute miliary tuberculosis, an extensive tuberculosis of the thoracic duct with breaking down of the tubercular new growth. It is easy to see how, in this way, a large amount of tuberculous material could be brought directly into the circulation, from the free communication of the lymph duct with the subclavian vein, and thus be "disseminated" through the different organs in a short time. Still more frequently, however, the tuberculosis of the large venous trunks, discovered by Weigert, especially the pulmonary veins, seems to be the starting-point for an acute general miliary tuberculosis. Usually there are tuberculous lymph-glands, or sometimes other foci of tuberculous disease, which involve the wall of a neighboring vein, gradually break through it, and project into its lumen. If caseation and ulceration result in this spot, the infectious material

is of course constantly washed off by the blood current and carried away, and thus it reaches the other organs.

Since such a tuberculous focus—e. g., a tuberculous bronchial gland—may remain for a long time entirely without symptoms, we can understand how miliary tuberculosis may break out in an acute form in persons who previously seemed perfectly well. In other cases the patient has already suffered from some tuberculous affection, and suddenly the conditions occur somewhere in the body which lead to the development of miliary tuberculosis. Thus we sometimes see it break out in a patient who has ordinary phthisis, but acute miliary tuberculosis is one of the rarities in advanced phthisis. If we find, at the autopsy of a case of acute general miliary tuberculosis, old phthisical changes in the lungs, which is by no means very common, they consist of old, partly cicatrized foci, pigment indurations, etc. We see miliary tuberculosis rather frequently as a sequel to pleuritic effusion. We have already called attention to the fact that in such cases the pleurisy itself is a tuberculous disease. Miliary tuberculosis is also seen in persons with old tuberculous affections of the bones and joints, such as coxitis and vertebral caries, with tuberculous swellings of the lymph-glands, as in the neck and the axillæ, or with tuberculosis of the genito-urinary organs, etc. In such cases, of course, the tuberculous affection which is discovered during life is not always the source of the general miliary tuberculosis, but the discovery of the existence of such an affection is of the greatest significance in diagnosis, as in this way our attention is strongly directed to the possibility of a general tuberculous affection.

In some cases an outbreak of miliary tuberculosis has been seen to follow other acute diseases, such as typhoid or measles.

Pathological Anatomy.—Except for the presence of an old tuberculous affection in some organ, and except for the tuberculosis of a vein or of the thoracic duct, which are usually demonstrable, and which have been described above, the anatomical lesion in acute miliary tuberculosis consists in the dissemination of miliary tubercles through a large number of the organs of the body. The lungs, the liver, and the spleen are constantly affected; almost as constantly the kidneys, the thyroid gland, the marrow of the bones, the heart, and the choroid; less constantly, but still quite frequently, the serous membranes and the meninges. The miliary nodules may be found in large numbers in all the organs mentioned. They may in part be easily recognized by the naked eye, and in the lungs they may be very plainly perceived by the touch. In many organs, however, especially in the liver and often in the spleen, they are hard to recognize with the naked eye, but they are easily discovered by the microscope. In regard to the histological structure of miliary tubercles, and the discovery of tubercle bacilli in them, we must refer to what has been said in the chapter on pulmonary tuberculosis, but we must also mention that, in some of the more chronic cases, some of the nodules may grow to be large tuberculous foci, from the size of a lentil to that of a pea. Less developed cases of miliary tuberculosis are also found, in which only a limited number of organs are attacked, and these with less severity.

Clinical History.—The clinical symptoms of miliary tuberculosis depend upon two factors, the first being the general infection of the body, and the second the local tuberculous affection of certain organs. Although in many organs miliary tuberculosis is entirely without symptoms, as in the liver, the

kidneys, the heart, and the marrow of the bones, in two organs—the lungs, and more especially the brain—it leads to the most marked local symptoms. The miliary tuberculosis of the choroid, discovered by Cohnheim and Manz, is also without symptoms, but it can be made out with the ophthalmoscope, and it is therefore of great diagnostic value.

Miliary tuberculosis affords quite different pictures, according to the predominance of one or the other of the groups of symptoms mentioned. We distinguish the four following forms:

1. *Miliary Tuberculosis, with Predominant Symptoms of General Infection: the so-called Typhoidal Form.*—This form may in part greatly resemble typhoid fever. The patient, who previously seemed quite well, or in whom some local manifestation of tuberculosis was suspected, falls ill with gradually increasing general symptoms, dullness, loss of appetite, headache, and fever. Since there is no local affection to be discovered to explain the symptoms, the disease at first may well be taken for typhoid. The general condition grows worse constantly, the fever is high and continually rises, and cerebral symptoms appear. In some cases an exanthematous eruption, like roseola, may increase the resemblance to typhoid. With careful observation, however, symptoms are almost always detected later in the disease which are, to a certain degree, characteristic of miliary tuberculosis, and are due to the existence of that disease either in the lungs or in the brain. The patient's complexion assumes a peculiar pallor, and with it a definite cyanotic hue. The respiration becomes remarkably deep, and there is dyspnœa; or signs of a tuberculous meningitis arise, such as rigidity of the neck, loss of consciousness, disturbances in the innervation of the ocular muscles, etc., and death follows with these symptoms. These cases last from ten days to three weeks, reckoning from the beginning of the severe symptoms.

2. *Miliary Tuberculosis, with Predominant Pulmonary Symptoms.*—These cases, too, may begin quite suddenly, almost like an acute croupous pneumonia, or they may develop gradually with quite a long prodromal stage. From the onset the symptoms point especially to disease of the lungs or the pleura. The patient complains of a stitch in the side, cough, and dyspnœa. The expectoration may bear a decided resemblance to that of pneumonia. Such cases, especially if they begin abruptly, are at first often erroneously regarded as croupous pneumonia, particularly if we find fine râles, almost like crepitant râles, over certain portions of the lungs. But the expected crisis does not occur: the fever continues; the dyspnœa, general weakness, and anæmia of the patient increase; the physical signs of pulmonary disease (diffuse bronchitic râles) become more and more extensive. The patient's face is pale, cyanotic, and anxious. Death ensues with all the signs of impaired respiration. The course is usually somewhat more protracted than in the typhoidal form, lasting for three or four weeks and more.

3. *Miliary Tuberculosis, with Predominant Cerebral Symptoms, due to Tuberculous Meningitis.*—Tuberculosis of the meninges does not belong among the regular lesions of general miliary tuberculosis. It develops in about half the cases, according to our estimation; but where it occurs it almost always gives the whole case the characteristic imprint of tuberculous meningitis, by which the other symptoms are entirely concealed. The predominant symptoms are headache, fever, stupor increasing to deep coma, rigidity of the back

and neck, and disturbances in the innervation of the ocular muscles. In such cases the tuberculous meningitis may alone be diagnosticated, and not the universal miliary tuberculosis. In fact, all the other signs of general miliary tuberculosis are not infrequently obscured by meningeal symptoms, and yet we have repeatedly observed, even in the deepest coma of the patient, a peculiarly deep and hurried respiration, which was the only noticeable sign referable to the miliary tuberculosis in the lungs.

The symptoms of tuberculous meningitis in many cases predominate in this type of the disease from the onset, but in other cases they come on during the attack and form its final period. The duration of the disease varies accordingly.

4. *Miliary Tuberculosis with a Protracted Course and Indefinite Symptoms for a Long Time—Intermitting Form.*—Besides the forms already mentioned, cases occur which usually take quite a protracted course, lasting for eight or ten weeks, and having such indefinite symptoms that an absolute diagnosis is for a long time, or even throughout the disease, quite impossible. The patient complains of a number of general symptoms, such as headache and dullness, and also of thoracic symptoms, for which, however, we can find on examination no sufficient basis. There is almost always fever, usually not very high, and with a very irregular course; but we have seen a regular daily rise of temperature for a time in some cases, and attacks of fever with quite a severe chill, so that at first we thought of an irregular malarial intermittent fever—the intermitting form. Later on the symptoms gradually increase. The apparently inexplicable loss of strength, and the patient's anæmia and emaciation, are marked, and they are important in diagnosis. Finally, either severe pulmonary symptoms or the signs of tuberculous meningitis set in, and to these the patient succumbs.

We must mention particularly that the four forms of miliary tuberculosis just described are only the types of the disease. In individual cases we often meet with variations and transitional forms between these types.

Single Symptoms.—1. *General Symptoms.*—In all cases of acute miliary tuberculosis the general condition of the patient is very serious. Most patients have a subjective feeling of severe illness, although they make little special complaint of it from the painless character of the disease. As the disease increases there is often a marked feeling of anxiety and oppression besides the dyspnœa. There is, especially in the face, quite a peculiar pallor, characteristic of the disease, and associated with a marked cyanosis of the lips and cheeks.

2. *Fever.*—Acute miliary tuberculosis almost always runs its course with a more or less high fever, a course without fever having been observed in only a few instances. It often happens, in more protracted cases, that the temperature may be nearly normal for a time, or only slightly elevated. There is nothing characteristic or typical in the course of the fever. In the cases with typhoidal symptoms the fever is usually quite high, between 103° and 105° F. (39.5° to 40.5° C.), so that the temperature curve may be exactly like that of typhoid. In other cases the fever is irregular and is broken by many remissions, remitting or intermitting quite regularly for some time. Death ensues with a moderately high temperature or in collapse. In cases with tuberculous

meningitis there is also a marked rise of temperature at the close, up to 108° F. (42° C.) and over.

3. *Respiratory Apparatus.*—It goes without saying that physical examination of the lungs may give no definite results. Almost all positive evidence is often wanting, and the contrast between the labored breathing and dyspnœa and the insignificance of the physical signs in the lungs is an important feature in diagnosis. Auscultation, as a rule, gives the signs of an intense bronchial catarrh; we hear rhonchi or numerous fine and medium moist râles all over both lungs. The respiratory murmur itself is usually higher in pitch than normal, and in many cases it is obscure, rough, or harsh. In our cases there was occasionally heard, over a circumscribed area of the lung, a wholly peculiar, sharp, "lapping" sound on inspiration. In quite a large number of cases peculiar fine crepitant râles are to be heard over some areas of the lungs; these, in our experience, are pathognomonic of miliary pulmonary tuberculosis. On percussion there are often no distinct objective changes to be found. With careful percussion, however, the abnormally deep note of the relaxed portions of the lungs, or a slight dullness over other areas, becomes very noticeable. We consider this varying quality of the pulmonary resonance (difference in pitch and the like) over different portions of the lungs of special diagnostic importance.

In some cases circumscribed pneumonic infiltration has been observed in the lungs in acute miliary tuberculosis, which, as we have said, may give rise to a confusion between miliary tuberculosis and croupous pneumonia, from the presence of marked dullness, crepitant râles, and bronchial respiration.

We must mention, finally, that in some of the cases physical examination of the lungs shows old changes in them, a phthisical affection of the apex, a former pleurisy, and the like. Positive evidence of such old tuberculous affections may be of great diagnostic value in doubtful cases.

Dyspnœa has been repeatedly mentioned among the pulmonary symptoms. The respiration is usually very much accelerated, especially during the more advanced stage of the disease, so that we see in adults forty, sixty, and even seventy respirations a minute. The respiration is also very deep, and is sometimes noisy. As a rule, there is cough, but it is usually troublesome only in the cases with severe bronchitis. It is often very slight. The expectoration is usually scanty, and it is not characteristic. Special mention must be made of the fact that tubercle bacilli are absent in it, unless old ulcerated tuberculous foci are present at the same time in the lungs.

4. *Circulatory Apparatus.*—The pulse is frequent, about 100 to 120 a minute, often weak and small, and sometimes irregular, especially if tuberculous meningitis coexists. The miliary tubercles, which postmortem are almost always to be found in the heart, especially in the endocardium, cause no symptoms. In uncomplicated, acute, miliary tuberculosis there is little if any increase in the number of white corpuscles in the blood. The presence of tubercle bacilli in the blood will be mentioned below.

5. *Digestive Apparatus.*—Vomiting is frequent at the onset of the disease. The bowels are usually constipated, but in many cases there is a moderate diarrhœa. The loss of appetite, the thirst, and the dry tongue are due to the general disease and the fever. The spleen is usually somewhat, but not very much, enlarged.

6. *Nervous System.*—In many cases in which the pulmonary symptoms predominate the intellect remains quite clear until the last, but in other cases cerebral symptoms, such as headache, dizziness, stupor, and delirium, come on quite early, and are part of the general infection. As has already been said, the nervous symptoms in the cases combined with tuberculous meningitis become quite prominent, but in individual instances it may be hard to decide whether they are due to such a complication, or are merely severe general symptoms.

7. *Eyes.*—The ophthalmoscopic examination of the retina is of special diagnostic importance, since the diagnosis may be made absolutely certain by finding miliary tubercles in the choroid. A negative result, however, is never decisive against the diagnosis, since the tubercles are sometimes absent, or at least are very few in number. Their discovery is almost always difficult, and demands much practice in the method of examination. In cases with tuberculous meningitis we sometimes find an optic neuritis.

Diagnosis.—The diagnosis of acute general miliary tuberculosis is ordinarily not very simple. It quite often happens that at the autopsy a miliary tuberculosis is found which was not even suspected during life. It must be confessed that frequently, in such cases, we might very well have thought of acute tuberculosis. If, therefore, the possibility of acute miliary tuberculosis is brought to our attention during the patient's life, we can occasionally make an absolute diagnosis.

The severe constitutional disturbance, usually associated with fever, is most important, and for this no local cause can be found. Then come the pulmonary symptoms, especially the peculiar dyspnœa, for which there is also no adequate corresponding physical change to be discovered. Besides the dyspnœa, a peculiar, cyanotic pallor is very characteristic. It gives decided support to our suspicion if we can make out a distinct predisposition to tuberculosis, either hereditary or constitutional, or the history of a previous tuberculous affection, especially pleurisy, and also chronic affections of the bones, and the like; or if any form of local tuberculous disease, as of the lymph-glands, a pulmonary apex, the pleura, etc., be present.

On these factors rests the differential diagnosis between the "typhoid" form of miliary tuberculosis and typhoid fever. Marked roseola and considerable enlargement of the spleen are distinct arguments for typhoid, although they sometimes occur in miliary tuberculosis, and so are the intestinal symptoms of typhoid, such as meteorism, the characteristic, loose yellow stools, and possibly intestinal hemorrhage; but we must not forget that both the roseola and the intestinal symptoms may be absent in typhoid. The course of the fever must always be considered in the differential diagnosis. It is much more frequently irregular and atypical in tuberculosis than in typhoid. Of course, the temperature curve is not an absolutely decisive factor. The proportionately slight acceleration of the pulse in typhoid is worthy of note. The blood does not afford any indubitable signs of distinction between the two, since in neither disease is there a decided leucocytosis. If, however, the number of leucocytes is conspicuously small, 5,000 or less, typhoid is strongly suggested. The Gruber-Widal serum reaction or the direct demonstration of the typhoid bacilli in the blood or in the dejecta (see page 25) is very important in the differential diagnosis; if positive, these tests are decisive.

On the other hand, of course, the absolute demonstration of miliary tuberculosis in the choroid is unequivocal evidence in favor of miliary tuberculosis. Weichselbaum and others have been able to demonstrate tubercle bacilli in the blood in a few cases. In general, however, this is difficult, and not always possible. [Ordinary and also distilled water sometimes contain acid-fast bacilli which may be mistaken for tubercle bacilli and cause errors in examining blood and feces.] In all doubtful cases the sputum must be very carefully searched for tubercle bacilli.

In many cases the onset of meningeal symptoms may aid the diagnosis. Of course, if the patient is not seen until the last stages of meningitis, especially when there is an incomplete history, the diagnosis is often very difficult. In this case, if the fluid obtained by lumbar puncture is found to contain tubercle bacilli, the diagnosis may be settled (compare the chapter on Tuberculous Meningitis in Vol. II).

Acute tuberculosis is often confounded with severe bronchitis, especially in old persons who are considered emphysematous. The very bad general condition, the pallor, the rapid loss of strength, and the fever will, when kept in mind, call our attention to acute tuberculosis, and render the diagnosis possible. We have already indicated the possibility of confusion at the onset between miliary tuberculosis and croupous pneumonia.

Prognosis.—The cases described in literature as "cured miliary tuberculosis" are so uncertain in their diagnosis that they cannot be regarded as convincing. We must therefore consider the prognosis is absolutely fatal. The differences in the course of the disease have been already mentioned.

Treatment.—Although drugs are absolutely powerless, still, the case in hand must always receive treatment, especially if the diagnosis cannot be made with absolute certainty. Our prescriptions are purely symptomatic. The cases with a typhoidal course are to be treated just like typhoid, with baths, stimulants, etc. Tepid baths, and also local applications to the chest, expectorants, and narcotics, are indicated when the thoracic symptoms predominate. If meningeal symptoms set in, we may try ice, perhaps local bloodletting, iodoform salve, or mercurial ointment externally, and iodid of potassium internally.

CHAPTER VIII

GANGRENE OF THE LUNGS

Ætiology.—The sole cause of pulmonary gangrene—that is, the death and putrid decomposition of the lung tissue—is the entrance of the bacteria of putrefaction into the lungs. The opportunity for inhaling them is certainly very great, but the normal organism apparently possesses the property of nullifying them and making them powerless. Under certain conditions, however, they take root in the lung and cause the death of the pulmonary parenchyma, which then, as a result of the presence of these specific bacteria of putrefaction, succumbs to that peculiar form of putrid decomposition known as "moist gangrene."

The factor which most frequently gives rise to the development of pulmonary gangrene is the entrance of organic foreign substances, especially bits of food, into the lungs. The bacteria of putrefaction either enter the lungs with the foreign substance, or they settle there later and set up a putrid decomposition, first in that portion of the lungs, and then in the neighboring lung tissue. The entrance of organic foreign substances into the lungs occurs in different ways. It often happens from swallowing the wrong way, or from an accidental inhalation. In this way pulmonary gangrene may arise in previously healthy people, but it occurs especially in patients who are very low, very stupid, and soporose, and also in the insane, in patients who cannot swallow or cough well, and in patients with paralysis of deglutition, as in bulbar paralysis. Bits of food may also reach the lungs from eructations and vomiting. Thus are explained the cases of pulmonary gangrene which occur in patients with cancer of the stomach, and, still more frequently, with cancer of the œsophagus. Putrid organic material may also reach the lungs from ulcerative and ichorous processes in the mouth, the pharynx, and the larynx. In cancer of the tongue, the pharynx, and the larynx, in other ulcerative processes, and in injuries or wounds from operations in the mouth and pharynx that have become septic, pulmonary gangrene may develop quite readily. Finally, septic foci in the vicinity may extend to the lungs or perforate into a bronchus. In this way pulmonary gangrene may arise from the perforation through the pleura into the lungs of an ulcerated cancer of the stomach or a gastric ulcer, or from the perforation of tuberculous bronchial lymph-glands into the œsophagus with the formation of an œsophago-bronchial fistula, etc.

In some cases the cause of the pulmonary gangrene cannot be made out, since the entrance of a foreign substance has perhaps been wholly unnoticed, as may happen in children, or during sleep. We had a grown-up young woman under observation for a long time with pulmonary gangrene, and one day she coughed up several fragments of chicken bones, but she could give no account of how they entered the lungs.

Experience teaches us that pulmonary gangrene is more apt to develop in persons with impaired nutrition, in old, marantic individuals and drunkards, than in those who are healthy. The tendency of patients with diabetes mellitus to pulmonary gangrene is remarkable.

Pulmonary gangrene often develops secondarily to some other pulmonary affection. We have already spoken of the relations between it and fetid bronchitis. Fetid bronchitis, on the one hand, often leads to pulmonary gangrene through an extension of the process to the alveoli; and, on the other hand, when there is a gangrenous focus in the lungs, the bronchi are often infected to a wide extent by the putrid secretion coming from it, and then there arises fetid bronchitis. Both diseases often run into each other without any sharp boundary; but gangrene may develop secondarily in other affections of the lungs. A new infection with putrid material, however, is always requisite, and the affection of the lungs that already exists furnishes merely a favorable soil. This is the only explanation of the process when croupous pneumonia "runs into gangrene," or when gangrene develops in catarrhal pneumonia, in bronchiectasis, or in phthisis.

Although the agents of putrefaction enter the lungs through the bronchi in most of the modes of origin of pulmonary gangrene that have been mentioned,

they may also be transported into the lungs by the blood current. We call these forms embolic gangrene. We find such gangrenous nodules in the lungs in connection with extensive gangrenous bedsores, puerperal processes, suppurative caries of the bones, etc. In these cases the putrid material enters a vein from the seat of the primary process and is brought to the lungs, and here we find, as a result of the putrid character of the embolus, not a simple infarction, but an embolic gangrene.

Pathological Anatomy.—We more frequently find pulmonary gangrene in the lower lobes than in the upper, corresponding to its mode of origin. Either both lungs are affected or only one, and the right somewhat more frequently than the left. We distinguish a diffuse and circumscribed form, according to the extent of the gangrene. Embolic gangrene belongs to the latter form, and its nodules, by preference, lie near the pleural surface.

We can easily recognize the anatomical changes in gangrene. The lung tissue is changed to a discolored, dirty, greenish-gray mass, which gradually and progressively becomes dissolved, forming a most foul-smelling ichor. We find, left in it, necrotic fragments of tissue and vessels. Gangrenous cavities, with irregular, ragged walls, are formed from the partial expectoration of the softened gangrenous nodule. The lung tissue in the vicinity of the gangrenous spot is to a greater or less extent inflamed, partly in the form of catarrhal pneumonia, partly in the form of circumscribed croupous pneumonia. The inflamed parts in the vicinity are gradually involved in the gangrene, so long as the process extends, but finally a suppurating line of demarcation may be formed about the gangrene, the whole gangrenous fragment is in a measure sequestrated, encapsuled, and gradually expelled, and so healing becomes possible. We have already stated that fetid bronchitis may arise from a gangrenous nodule.

Whenever a gangrenous nodule reaches the pleura, a purulent and usually a sanious pleurisy follows from direct infection. Pneumothorax may arise from perforation of a gangrenous cavity.

Clinical History.—The symptoms of gangrene depend for the most part upon the local affection in the lung. The condition of the expectoration is characteristic, and it alone may decide the diagnosis.

In many ways the expectoration greatly resembles that of fetid bronchitis, and indeed a great part of it does not come directly from the gangrenous nodule, but is the secretion of the diseased bronchi. The penetrating stench of the sputum, a most repulsive, putrid odor, is very striking. The patient's breath and cough also have this stench, which infects the whole vicinity. The amount of the sputum is usually large; it may reach 8 or 16 ounces (200 to 500 c.c.) in twenty-four hours. If the sputum is collected in a glass it forms three layers, like the sputum of fetid bronchitis—an upper layer, mucopurulent, greasy, consisting in part of nummular sputa, and covered with much froth; a middle serous layer, in which some firm masses from the upper layer float; and a lower layer, almost wholly of pus, but greasy and greenish-yellow, which usually contains many large and small plugs and shreds of tissue. We find in these plugs, on microscopic examination, beautifully twisted needles of the fat acids (see Fig. 40, page 203) imbedded in countless bacteria, fat-drops, and detritus, and often collected in large bundles; but besides these we find in the sputum the constituents of the parenchyma of the lungs,

and this alone is the decisive factor in distinguishing between pulmonary gangrene and simple fetid bronchitis.

Traube's statement that in pulmonary gangrene the expectoration contains few, if any, elastic fibers, because the elastic tissue itself is destroyed by gangrene, is not correct; or, at any rate, it is too sweeping. We have almost invariably found in the expectoration an abundance of elastic tissue, as well as other fragments of the parenchyma, pigment granules, and the like. Yet there is a probability, no doubt, that in pulmonary gangrene the elastic tissue is for the most part destroyed. Filehne succeeded in extracting from the sputum of pulmonary gangrene, by means of glycerin, a ferment which in alkaline solution completely dissolved elastic tissue in a few days. The sputum always contains in enormous numbers bacteria of various kinds, both cocci and bacilli. Which of these are the special cause of the gangrene has not yet been definitely settled. The chemical examination of the sputum shows the presence of those substances which may always be found in the putrefaction of organic matter—tyrosin, leucin, ammonia, sulphureted hydrogen, butyric acid, valerianic acid, caprylic acid, etc. The fresh sputum usually has an alkaline reaction, but on standing it becomes acid.

Many cases of gangrene lead to erosion of the vessels and severe hemoptysis. Slight admixtures of blood in the sputum are not infrequent.

The other symptoms on the part of the lungs are not especially characteristic of gangrene. Most patients complain of cough, pain in the side, and more or less severe dyspnœa. Physical examination, as a rule, permits us to make out the seat of the nodule, but not always, since the physical signs, of course, depend upon the situation and extent of the gangrene. Small nodules, situated centrally, often give no objective evidence of their presence. Every extensive infiltration, however, must cause dullness on percussion. Over the area of dullness we hear bronchial respiration, and usually quite numerous moist râles. If a gangrenous cavity is formed, the physical examination may show plain symptoms of a cavity—tympanitic resonance on percussion, amphoric respiration, coarse moist râles, etc.

The physical signs are sometimes due to the accompanying pleurisy; the dullness is more complete, the respiratory murmur and the vocal fremitus are diminished, and the adjacent organs are displaced by the abundant effusion; but an absolute diagnosis of an accompanying pleurisy is often to be made only by an exploratory puncture. We have already spoken of the occasional development of pneumothorax.

In many cases there is fever, of quite an irregular character and of very varying intensity. In the cases in which the gangrenous nodule is sequestrated, and the secretion can be freely emptied through the bronchi, so that there is no absorption of septic material into the blood, fever may be entirely absent.

We often see gastric and intestinal symptoms in pulmonary gangrene, the disturbance being without doubt due to swallowing some of the fetid sputum. Many patients complain of loss of appetite, and sometimes of vomiting or diarrhea. In severe acute cases there is sometimes a well-marked typhoidal state, with such symptoms as stupor, delirium, and great cardiac weakness. This condition is probably caused by the absorption of septic material into the blood. Rheumatic pains in the muscles and joints are seen in this disease,

just as in fetid bronchitis. Finally, it should be remarked that the appearance of secondary abscesses in the brain (see Vol. II) has been repeatedly observed in pulmonary gangrene. This fact must be borne in mind when, in the course of pulmonary gangrene, marked brain symptoms are developed, especially if we have not only such general symptoms as coma, but also such local symptoms as hemiplegia and other forms of paralysis, and convulsions.

The general course of the disease shows very great variations. In all cases in which the pulmonary gangrene is secondary to some other affection, the course and the general type of the disease depend very largely upon the primary attack, but the cases of idiopathic gangrene also present great variations. The onset of the disease is either quite gradual and slow, or quite acute, and associated with fever and thoracic symptoms. The stinking expectoration and the bad odor from the patient's mouth first direct the attention to the existence of putrid processes in the lungs. The disease is usually very chronic, lasting for months or even years. Many remissions and intermissions occur. With proper care and treatment we may see a decided improvement, and often apparently a complete cessation of the disease. The bad odor ceases, the expectoration diminishes or disappears entirely, and the patient's strength and nutrition become almost normal; but relapses may occur after long intervals. When the affection is of slight extent we may even see a complete recovery.

Pulmonary gangrene always takes a worse course in previously weak and marantic persons, and an unfavorable termination may follow in a comparatively short time. Death ensues either from a general loss of strength, as a result of the disease, or from complications, such as pulmonary hemorrhage, ichorous pleurisy, pneumothorax, or abscess of the brain. Rupture of an ichorous empyema outwardly, or into the peritoneum, or into other cavities, is rare.

Special mention must be made of the fact that the symptoms of pulmonary gangrene are not always so very pronounced. In persons who are weak and run down we often see pulmonary gangrene at the autopsy, although during life there have been no marked symptoms, not even offensive sputum or fetor from the mouth.

Diagnosis.—The diagnosis cannot be made with certainty unless the characteristic sputum is present. We can decide whether the sputum comes from a fetid bronchitis or from the fetid contents of a bronchiectasis, or from actual gangrene, only by finding under the microscope the remains of lung tissue in the expectoration. Physical examination in gangrene, at least in part of the cases, also gives the signs of infiltration or of cavity formation in the lungs. In addition to the ordinary methods of physical examination, X-ray examination is of great importance.

Prognosis.—The prognosis depends first upon the nature of the underlying disease, and then upon the extent of the affection, the strength of the patient, and the possibility of sufficient care and proper treatment. If the process in the lung becomes sequestrated, marked improvement may follow, even in the severest conditions; but we must always remember that a relapse is possible. Complete recovery from pulmonary gangrene is certainly extremely rare, if it ever occurs. We have already mentioned the dangers which may lead to a fatal termination.

Treatment.—Prophylaxis plays an important part in those cases in which there is danger of the entrance of bits of food into the air-passages from defective deglutition. We must think of the possibility of this with all patients who show great stupor, and also with patients who have pharyngeal paralysis, in order to watch them while taking food, and eventually to try artificial feeding with an œsophageal tube.

The treatment of already existing pulmonary gangrene has, as its chief aim, to check the putrid processes of decomposition in the lungs. Unfortunately, the remedies at our command are not in all cases sufficient for this. The different disinfecting inhalations are the most effective. They are used in the same way as in fetid bronchitis (*vide supra*). Turpentine deserves the most confidence, and it may also be given internally with good results. According to Lépine, however, terpin hydrate acts even better than turpentine. We may also call attention to inhalations with carbolic acid, Curschmann's carbolic mask, inhalations with salicylic and boric acids (4 parts of salicylic and 20 of boric acid to 1,200 of distilled water), bromin (bromin and bromid of potassium, 2 parts of each to 1,000 of water), and similar remedies.

Besides oil of turpentine, other internal remedies are recommended: 0.5 to 1 gr. (gm. 0.03 to 0.06) of acetate of lead every two hours, creosote, guaiacol, etc. Their action is uncertain. Of late, myrtol has been greatly extolled. It is given in capsules containing gr. ijss. (gm. 0.15), of which two or three are to be taken every two hours.

The general treatment of the patient is very important—he should have good food, and live in as good air as possible. We must treat the pain in the chest and the cough symptomatically, local applications and morphin being most useful. The fever seldom gives occasion for direct interference. We may try to relieve the gastric and intestinal symptoms by giving antiseptics internally, especially by small doses of muriatic acid, salicylic acid, or creosote, as well as by the ordinary remedies, such as bitters and opium.

If a secondary ichorous pleurisy develops, with or without pneumothorax, removal of the fluid by operation is necessary, if the patient has sufficient strength to bear it. Direct incision and drainage of the gangrenous area in the lung has also recently been tried. It is an operative procedure that is at least worthy of further trial and development. Lenhartz, of Hamburg, in particular, has reported some encouraging results from operative intervention.

CHAPTER IX

DISEASES FROM THE INHALATION OF DUST

(Pneumonoconiosis)

Although there are a number of important contrivances in the respiratory apparatus to prevent the entrance of foreign substances into the lungs, still, if a person remains in a dusty atmosphere, so many particles of dust may be inhaled that they are not without effect on the lung tissue. The diseases arising from the inhalation of dust are usually purely vocational diseases, which occur especially in workmen whose occupation involves the con-

tinual inhalation of some kind of dust. In earlier chapters of this book, particularly while considering chronic bronchitis, we have already emphasized the harmful influence of the inhalation of dust. We have seen how the habitual respiration of organic dust in particular (from grain, wool, wood, and tobacco) is very apt to lead to severe forms of bronchitis and bronchiolitis. We must now draw attention to certain specific diseases similarly caused.

We must first mention, however, a condition of the lungs which can scarcely be regarded as pathological, although it has its origin in the constant inhalation of dust, especially of coal dust—the ordinary black pigmentation of the lungs. There can now no longer be any doubt, although there was once a long dispute about it, that the black pigment in the lung comes, in large part, at least, from the inhalation of carbon. The particles of carbon pass into the lungs themselves, and thence into the bronchial glands by means of the lymphatics. A certain part of the coal dust inhaled is removed with the expectoration, and it may easily be found in it microscopically, and often by the naked eye, as we see it in the well-known black expectoration which we often have in the morning, if we have spent the previous evening in a room filled with smoke. In Germany, Traube was the first to discover the particles of carbon in the expectoration of a charcoal burner. In the man's lungs, after death, the vegetable origin of these particles could be recognized, and Traube gave the correct explanation of them. In workmen who inhale large amounts of charcoal dust, anthracite coal dust, soot, or graphite, the "normal" pigmentation of the lung passes into a pathological condition, *anthracosis pulmonum*. With this is usually associated an extensive chronic bronchitis. In the expectoration of such patients are found many cells filled with black particles of coal, even long after they have left the dusty atmosphere. The pigmented cells are leucocytes, or perhaps some of them epithelial cells (see Fig. 56).

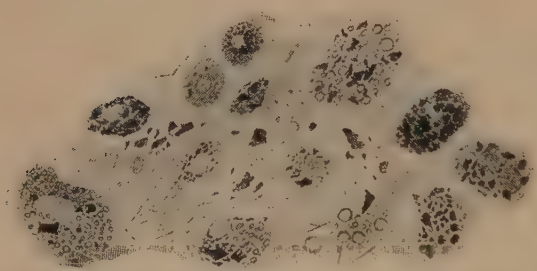

FIG. 56.—Expectoration of a man who worked on graphite. Numerous cells filled with particles of carbon. (Erlangen Medical Clinic.)

Zenker first discussed comprehensively the fact of the entrance of the different sorts of dust into the lungs and the consequent results. Besides the anthracosis already mentioned, the pulmonary disease from inhaling the dust of flint and other stones is of especial importance, the so-called stonecutter's lung—*chalicosis pulmonum*—and also that from inhaling metallic dust, especially oxid of iron—*siderosis pulmonum*. The "stone lungs" have been observed in workmen in the stamping rooms of glass factories, in millstone cutters, stone polishers, stone hammerers, plasterers, workers in porcelain, masons, slate quarrymen, potters, etc. "Metal-dust lungs" occur in file cutters, iron workers, mirror polishers, and especially in grinders, who inhale a mixture of stone and iron dust. The first case of a "red-iron lung" was observed by Zenker, in a girl who had inhaled a thick dust of iron for ten

or twelve hours a day. Her work was to color blotting paper with a powder of red oxid of iron. During the inhalation of all these and similar kinds of dust, a portion of the inspired particles of dust are taken up by the leucocytes, and perhaps also by the epithelial cells, finally reaching the lymph-channels of the lungs. Some of the particles of dust remain in the interstitial connective tissue of the lungs. Others find their way to the bronchial and retro-bronchial lymph-glands.

If the inhalation of dust is long continued, it causes not only this abnormal pigmentation of the lungs, but also macroscopic anatomical lesions. These consist, partly, in a more or less severe and extensive chronic bronchitis, and partly in an interstitial inflammation which is due to the chronic irritation of the foreign matter (e. g., flinty particles), and leads to the formation of connective tissue. The lungs are studded with nodules, which feel hard to the touch, and grate on section with a knife. All of these nodules consist of firm connective tissue, in which the particles of stone or iron are encapsuled. By the union of single nodules we may get more extensive induration and cicatricial formation. Chemical examination of such lungs gives, as might be supposed, a large amount of silicic acid, iron, etc.

In most of the cases which come to autopsy we find further changes in the lungs, which are not the immediate result of the inhalation of dust, but consist of sequelæ and complications. Chronic diffuse bronchitis in the worker in dust, like any other chronic bronchitis, may give rise to pulmonary emphysema, and later to cardiac hypertrophy, etc. We very often find in the lungs co-existing and pronounced tuberculous changes. It need hardly be emphasized that these changes are not the direct result of the inhalation of dust, but that the changes in the lungs set up by such an inhalation furnish merely a favorable soil for infection with tuberculosis. In most cases, the "dust lungs" acquire a marked clinical significance chiefly from the sequelæ mentioned—namely, emphysema and tuberculosis. The circumscribed nodules of interstitial pneumonia have no very marked symptoms following them. In all cases in which there is a fatal termination, with pulmonary symptoms, we should regard the immediate action of the dust as much less the cause of death than are the secondary diseases.

The essential points to be considered in judging of the clinical symptoms of the diseases from inhaling dust are contained in what has been said. The symptoms are those of chronic bronchitis, pulmonary emphysema, or chronic phthisis, and attention to the injurious influences associated with the patient's calling is the only possible way of making a diagnosis, but in individual cases it may always be a matter of doubt how far other accidental causes of disease may come into play.

The prognosis depends, in the first place, upon whether the patient can be removed from the action of these injurious influences or not, but we must also mention the fact, which has been often observed, that many individuals get somewhat used to the dust. After they have once recovered from the initial bronchitis, such persons can live in an atmosphere of dust for a long time without any noticeable injury.

The prophylaxis of diseases from inhaling dust forms an extended chapter in the hygiene of occupations, which we cannot dwell upon here. The workman must be taught the danger to which he is exposed, and the danger itself

must be diminished as much as possible by sufficient ventilation of the work-rooms, by cleanliness, and, under some circumstances, by a change in the technicalities of the business.

We need not give any special directions regarding the treatment of diseases from inhaling dust. It is founded on the same principles as the treatment of chronic bronchitis, emphysema, and chronic pulmonary tuberculosis.

CHAPTER X

EMBOLIC PROCESSES IN THE LUNGS

(Hemorrhagic Infarction of the Lungs)

Ætiology.—The sources from which the material for an embolic plugging of branches of the pulmonary artery comes lie either in the right side of the heart or in the veins of the body. Pathological anatomy teaches us how often thrombi are formed in the veins, especially in the veins of the lower extremities and in the pelvic veins, and in the right side of the heart, in the recesses between the trabeculæ, in the auricles, on the valves and the chordæ tendinæ, and at the apex of the ventricle. The particles torn loose from a thrombi so situated are carried on by the blood current, reach the lungs, plug a larger or a smaller branch of the pulmonary artery, according to the size of the particles, and thus cause further changes in the lung tissue. Since the branches of the pulmonary artery are "terminal arteries," and since thus the vascular territory belonging to each branch cannot be supplied, or can be supplied only to a small amount, with blood by collateral circulation from other vessels, the closure of a branch of the artery shuts the territory supplied by it out of the circulation. The pressure, in the part of the vessel lying peripherally to the point of plugging, becomes almost nil, and, as a result, there is a collateral or backward current into the region shut off, flowing from the capillaries in the vicinity, and probably even from the veins belonging to it. Yet the blood which flows in is under so slight a pressure that it does not flow through, but stagnates in the affected area. The walls of the capillaries and veins, in which the normal blood current has ceased, lose their natural consistence as a result of this. The vascular walls become abnormally pervious. The fluid of the blood, the white blood corpuscles, and also very many red blood corpuscles, penetrate through the walls of the vessel into the surrounding tissue, and change it into the so-called hemorrhagic infarction. All these changes develop more readily if the pulmonary vessels have already been subjected to chronic stasis. For this reason, pulmonary infarcts are very apt to develop in cases of cardiac disease (especially in mitral stenosis), whereas we quite frequently find, especially in central portions of the lung, embolism of single branches of the pulmonary artery without the formation of an infarction. In such cases there must necessarily be a little circulation in the vascular territory which has been shut off, either by anastomoses between the territory of the pulmonary artery and that of the bronchial or mediastinal artery, or by the neighboring capillaries, whose arteries of supply remain open. Death may immediately follow the sudden embolic obturation of a main trunk or of several of the larger

branches of the pulmonary artery; thus further changes in the pulmonary tissues may not have time to develop.

The changes thus far described are the result of a purely mechanical closure of a pulmonary artery. We have noticed this especially when simple fibrinous plugs have given the occasion for the embolic process. Pulmonary infarctions are most frequent in chronic heart disease, in all forms of primary and secondary dilatation of the heart, but especially in disease of the mitral orifice, most often mitral stenosis. Thrombus formation is frequent in the dilated right side of the heart, and furnishes material for pulmonary emboli; but these emboli are seen in all other possible conditions of disease, in which thrombosis in the right side of the heart or in the veins may occur (varices of the lower extremities, etc.). Numerous observations in recent years have taught me the great importance and frequency of pulmonary embolism as a sequel to various operations (especially laparotomies), and to confinements. We need not discuss the ease with which the smaller veins may become thrombosed under these circumstances, and the likelihood of a pulmonary embolism from such a source. Very many cases of pneumonia and of pleurisy developing after major operations or during the puerperium are unquestionably of embolic origin.

The question as to whether the embolic material is composed of fibrin alone, or of fibrin combined with special infectious substances, is one of considerable importance. Inasmuch as that portion of the lung thrown out of circulation by an infarct offers no opposition to invasion by bacteria, a simple infarct may also be followed by inflammatory changes in the pulmonary tissue or in the neighboring pleura. Embolic inflammatory processes occur more promptly, however, when the embolus itself is of an infectious nature.

If, for example, emboli reach the lungs from an acute malignant endocarditis in the right side of the heart, or, as is most frequently the case, from a purulent (septic) phlebitis anywhere in the body, giving rise to a puriform, liquefying thrombus, the specific factors in inflammation—that is, bacteria—get into the lungs. Thus arise embolic abscesses and embolic gangrenous nodules in the lungs. We have already spoken of the latter, and the former are among the most constant lesions in every typical case of pyæmia.

The fundamental facts as to the occurrence and significance of embolic processes in general, and those located in the lungs in particular, were discovered by Virchow. For a fuller understanding of the results of embolic closure of the vessels we must thank chiefly Cohnheim.

Pathological Anatomy.—Hemorrhagic infarctions may involve one or more lobules, or almost a whole lobe of the lung, according to the situation of the embolus. Most infarctions are situated at the periphery of the lung, and have approximately a conical shape, corresponding to the extent of the region of the vessel. The base of the cone lies against the surface of the pleura. It generally projects a little above that surface, and its dark color can usually be plainly recognized through it. The pleura itself is the seat of a fibrinous inflammation at the point to which the infarction reaches, and sometimes for a large space around it. The conical shape of the infarction is plainly recognized on section. The lung tissue is changed to a solid, fragile, uniformly black-red tissue devoid of air. The embolus can usually be readily

found in the branch of the pulmonary artery leading to the infarction. Under the microscope we see a diffuse infiltration of tissue, with red blood corpuscles in the infarcted portion. The alveoli and finer bronchi are also filled with coagulated blood. Under favorable circumstances, in cases of longer standing, the blood may be reabsorbed in part; so that the lung again contains air, but it remains much pigmented in that place, and more or less indurated from the development of interstitial connective tissue. In other cases various inflammatory changes result from the infarction (embolic pneumonia, embolic pleurisy). Destruction and absorption of the infarcted tissue with secondary formation of scar tissue is, in contrast to other organs, only rarely observed in the lungs.

Hemorrhage infarctions are almost always found in the lower lobes, sometimes in the right middle lobe, rarely in the upper lobes. The embolic abscesses are sometimes very numerous, and are disseminated over the whole lung. In larger abscesses the conical shape may often be plainly recognized. When an abscess extends to the pleura, a purulent pleurisy arises from direct infection. Combinations and transitional forms between the ordinary hemorrhagic infarction and embolic abscesses are occasionally found in the lungs.

Symptoms.—We often find at the autopsy embolism of single branches of the pulmonary artery, with or without infarction, which has caused no symptoms at all during life.

Embolism of the main trunk, or of a large branch of the pulmonary artery, may cause sudden death, as has not infrequently been observed in patients with heart disease, or with venous thrombosis (sudden death in cases of thrombosis of the femoral vein, after operations, etc.). If death be not immediate, sudden severe dyspnœa and a sense of oppression occur. The patients become pallid, cyanosed, and pulseless; consciousness is lost, and death ensues in the briefest space of time, often ushered in with slight convulsive twitchings. The diagnosis may at least be suspected in such a case if we know of a possible source for an embolus. In some instances, when an embolus is situated in a large branch of the pulmonary artery, but has not completely filled it, we can hear a systolic vascular murmur over the affected spot, as has been observed by Litten.

If we are dealing with cases of nonfatal embolism of the smaller vessels, the first result of the embolism is usually a sudden onset of severe pain in the side, combined with interference with respiration and a sense of oppression. In cases of centrally situated infarctions, pain may be absent, since it probably is dependent on pleural involvement. The characteristic bloody sputum seen in many cases usually appears at a somewhat later date. Either the sputum consists almost entirely of dark blood, or the blood is mixed with more or less mucus; but there is never much air in it. The bloody expectoration often lasts for several days. It is sometimes absent altogether, despite existing infarction; or, when no marked infarction of the tissues has developed, it may be only slightly or not at all blood-tinged. In a few cases of pulmonary infarctions we have noticed an exceedingly large number of cast-off alveolar epithelial cells in the sputum.

We try to learn more of the size and situation of the lesions by a physical examination of the lungs. Of course this often gives a negative, or at least a doubtful, result. It goes without saying that small infarctions, and also

those which are central, cannot be made out by physical examination. Large peripheral infarctions may give rise in many cases to dullness on percussion, crepitant râles, and harsh or bronchial respiration, but it is often hard to decide in an individual case whether the physical signs which we meet with are not due to other pathological changes, such as bronchitis, pleurisy, or hydrothorax. We sometimes hear a pleuritic friction-sound in some part of the chest a few days after we suspect that an infarction has occurred, by which the diagnosis gains additional certainty.

Fever may be wholly absent, though we sometimes see a moderate rise of temperature at the onset. Fever often appears one or two days after the onset of the subjective symptoms; a circumstance that appears to us to be specially characteristic of embolic inflammation. Embolism occasions pain and a sense of oppression; the subsequent secondary inflammation (*vide supra*) causes the fever.

The embolic abscesses in the lungs hardly ever give rise directly to clinical symptoms. They form a part of the general picture of pyæmia and similar general infectious processes. Marked symptoms on the part of the respiratory apparatus are seen only when the abscesses are present in very large number. If an empyema develops from a focus which extends to the pleura, it sometimes occasions definite physical signs.

It follows from all that has been said, that in the diagnosis of embolic processes the chief stress must always be laid on the presence of an ætiological factor. We must regard the bloody sputum as the main direct symptom in hemorrhagic infarction. Embolic abscesses in the lungs may often be suspected in pyæmic diseases, but they can hardly ever be diagnosticated with certainty.

The prognosis is entirely dependent upon the underlying disease. In heart disease the occurrence of a hemorrhagic infarction is usually on the whole an unfavorable sign, since it points to weakness of the right ventricle, occasionally the formation of a thrombus in it; yet it often happens that the symptoms of a pulmonary infarction may pass away entirely.

We need not give special directions for treatment. It is in part purely symptomatic, and in part coincident with treatment of the underlying affection. As regards prophylaxis, we must bear in mind the absolute necessity of as perfect rest as possible for those patients in whom the presence of venous thrombi—e. g., in the femoral veins—suggests the possibility of pulmonary embolism.

CHAPTER XI

BROWN INDURATION OF THE LUNGS

(Lungs of Heart Disease)

In heart disease, especially in mitral stenosis, we often find a peculiar change in the lungs, whose origin must be sought in the long-persisting engorgement of the pulmonary circulation. The lungs are hard and dense, and show on a fresh section an abnormal brownish-yellow color. In the larger pulmonary vessels, both arteries and veins, there is a thickening and cloudi-

ness of the intima as a result of the stasis. We see here and there on the surface of the section, and beneath the pleura, little dark spots of pigment and fresh hemorrhages. We term this condition brown induration of the lungs.

Microscopic examination shows that the capillaries are evidently dilated and twisted as a result of the persistent stasis. They even extend a good way into the alveoli, whose lumen is thus actually diminished. The interstitial connective tissue seems somewhat thickened, and we find in it many brown pigment granules, the remains of the extravasated and decomposed red blood corpuscles. The pigment granules are some of them free, and some contained in cells. In the intima of the larger vessels we often find fatty degeneration of the endothelium.

With regard to the clinical importance of the pulmonary changes due to heart disease, we should say that it is very probable that the dyspnœa of such patients is aggravated by the diminution of the alveolar spaces throughout the lungs, as a result of their being crowded with desquamated pulmonary epithelium. Clinically, however, we cannot well distinguish this factor from the other causes of dyspnœa.

We have no positive factors by which to diagnosticate brown induration of the lungs during life. The anatomical lesions, too, show a certain variation, not always to be explained, in that, under apparently the same conditions, the brown induration is often very marked, and often extremely slight. In cases in which we find this induration in the cadaver we have repeatedly heard, during the patient's life, a very sharp, puerile respiratory murmur, which seems to be characteristic of many cases of the "heart-disease lung." In the diagnosis of "heart-disease lung," I place the greatest weight on the ofttimes highly characteristic sputum. Even on macroscopic examination, the latter often presents an appearance peculiarly its own. It is rather tenacious, mucoid sputum containing scarcely any pus cells, and showing a large number of areas that have a dirty brown, coffee-colored appearance. If the brown spots be examined under the microscope, their color will be seen to be due to numerous large cells which are closely filled with smaller and larger, yellow to brown-colored pigment granules (see Fig. 57). These large pigmented cells ("cells of heart disease") are identical with the above-mentioned pigmented cells found on the interior of the alveoli upon anatomical examination of the lungs. They are certainly for the most part alveolar epithelium filled with pigment; some, however, are probably leucocytes that have taken up the pigment from degenerated red blood cells. When the sputum is treated with hydrochloric acid and potassium ferrocyanid, a distinct blue color appears.

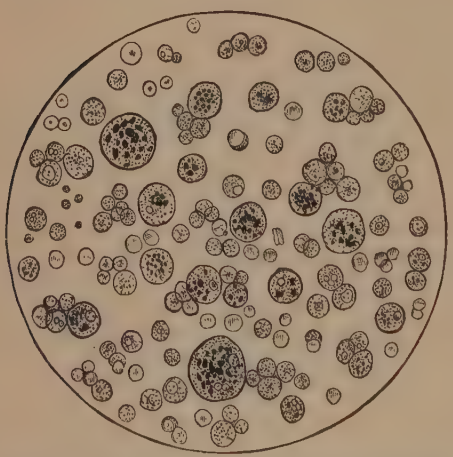

FIG. 57.—Sputum of a patient with mitral stenosis, containing the so-called "cells of heart disease." (Personal observation.)

This test for iron may be macroscopically performed in the sputum glass, or microscopically, on the stage of the microscope. Besides the pigment cells, a remarkably large number of "myelin drops" as well as unaltered red blood cells are usually to be seen.

CHAPTER XII

TUMORS OF THE LUNGS. CANCER OF THE LUNGS. ECHINOCOCCUS OF THE LUNGS. PULMONARY SYPHILIS

1. NEW GROWTHS IN THE LUNGS. CANCER OF THE LUNGS

Secondary New Growths.—Most of the new growths which are met with in the lungs are of a secondary nature. Secondary cancers are sometimes found in the lungs, with carcinoma of other organs, whose origin may almost always be explained by supposing a growth of the primary tumor into a vein, and the consequent carriage of the germs of the growth to the lungs. In other cases, the tumor material reaches the lungs by way of the lymph-channels. In such cases the spread of the tumor not infrequently occurs in the form of strands along the axes of the lymph-vessels. These secondary nodules in the lungs often cause no special clinical symptoms, unless they are very numerous and extensive, when they give rise to dyspnœa and physical signs. There once came to the clinic in Leipsic a case of secondary, and very extensive, miliary carcinosis of the lungs, which ran a brief and fatal course, simulating acute miliary tuberculosis with predominant pulmonary symptoms.

Other secondary new growths deserving mention are enchondroma and sarcoma. We have seen an extensive development of secondary pulmonary sarcoma, following primary sarcoma of the bronchial lymph-glands, and also in another case following lympho-sarcoma of the lymph-glands of the neck, which grew into the jugular vein. We have also seen secondary deposits of sarcoma repeatedly in connection with primary congenital sarcoma of the kidney (*q. v.*).

Primary Cancer.—Of the primary new growths of the lungs, only pulmonary or bronchial carcinomata have any great clinical significance. All other new growths (enchondroma, sarcoma, fibroma, osteoma, etc.) are rare, and possess scarcely any but an anatomical interest. Primary cancer of the lungs usually originates in the epithelium of the bronchial wall, though sometimes it originates in that of the alveoli. It is especially common in elderly persons, over forty, and seems to be found somewhat more frequently in the right lung than in the left, and in the upper lobes than in the lower. By its diffusion the lung tissue in the parts affected by cancer is changed to a yellowish-gray and quite soft and crumbling mass, devoid of air. We can usually scrape away from the section the characteristic cancer juice, in which the microscope shows the typical cancerous elements. The pleura is very often involved. The new growth has either extended directly into it, or single, and more circumscribed, secondary nodules have formed in it. The lymph-glands are almost invariably affected, especially the bronchial glands, and also the axillary and cervical glands. Secondary carcinoma of other organs is rare, but it is found in some cases in the other lung, the liver, the brain, and elsewhere.

It is almost always difficult to correctly interpret the clinical symptoms of cancer of the lungs at the beginning of the disease. They are referred to some other, more common chronic pulmonary disease, such as chronic bronchitis, phthisis, or pleurisy, but in the further course of the disease we succeed, at least in a number of cases, in making a correct diagnosis. In other cases, especially in old people, the growth may remain latent.

The general pulmonary symptoms have little that is characteristic. The patient complains of gradually increasing difficulty in respiration, and of pressure and distress in the chest, which may finally increase to the most intense dyspnœa. Most patients suffer very much from the labored, frequent, and spasmodic cough. The expectoration in some cases has no peculiarity, but it often assumes, at least for a time, a characteristic consistence which is extremely important for diagnosis. The presence of blood in the sputum is of the greatest importance. Repeated slight or even considerable hemoptyses are often the first symptom to call attention to the beginning of serious disease. In some cases the expectoration becomes permanently hemorrhagic, and then it not infrequently assumes a peculiar " raspberry-jelly " appearance. Under the microscope we can sometimes make out the characteristic elements of the tumor in it.

Physical examination frequently gives no positive results at the outset. In the cases observed by the author, a striking diminution of the respiratory murmur over localized areas, despite normal pulmonary resonance, was often the first decided change in the physical signs. It was due to the increased obstruction of the afferent bronchus by the growing tumor. A stridor is also occasionally heard when the bronchial lumen is encroached upon. After the tumor has extended, naturally more distinct signs are to be found (dullness, bronchial breathing, râles, pleuritic friction sounds, etc.). Frequently the peculiar situation of the areas of dullness (in the sternal region, for example) of itself leads one to think of a new growth. As soon as the pleura becomes involved, the sense of resistance upon percussion is very marked. Not infrequently a diffuse protrusion of the chest wall, in the region of the disease, appears. Sometimes there is also a slight œdematous swelling of the skin. The X-ray examination of the lungs is of almost greater importance than percussion and auscultation. It permits of the distinct recognition of deep-seated tumors, and gives the best indication of the extent of the disease. It should, therefore, never be neglected in any case of moment.

The occurrence of certain sequelæ is of great diagnostic significance. The chief one is swelling of the lymph-glands in the neck or axilla, and also certain symptoms of compression, which either are produced directly by the new growth, or are due to the secondary enlargement of the lymph-glands. Pressure on the superior vena cava, or a large branch of it, causes œdema in the face, neck, over the wall of the chest, or in one arm. The subcutaneous veins in the regions named appear dilated and tortuous. Pressure on the œsophagus causes difficulty in deglutition; on the brachial plexus, intense neuralgic pains and paresis of one arm; on the recurrent nerve, paralysis of the vocal cords and hoarseness; on the trachea or a primary bronchus, symptoms of tracheal or bronchial stenosis. The pleura is often involved so that the signs of a pleuritic effusion associate themselves with the other symptoms. In such cases the exudation is not infrequently hemorrhagic (see new growths of the pleura).

Besides the symptoms already mentioned we must consider the constitu-

tional symptoms. As in carcinoma in general, so in pulmonary carcinoma, the well-known cancerous cachexia gradually develops. The patient grows dull, loses his appetite more and more, disturbances of digestion and sometimes moderate elevations of temperature develop, until he finally succumbs to general marasmus.

The whole duration of the disease is from six months to two years. The prognosis is fatal. The treatment can be only symptomatic, and we employ the same remedies as in other pulmonary affections.[1] X-ray treatment or subcutaneous injections of arsenic may be tried in cases of sarcoma or lympho-sarcoma of the lungs. Palliative results, at least, are sometimes attained by these means.

Lympho-sarcomata of the Lungs.—We must still briefly consider a new growth in the lungs which is extremely interesting from a theoretical point of view. In workmen in the cobalt mines of Schneeberg, in the Saxon Voigtland, the development of malignant lympho-sarcomata in the lungs, with the occasional formation of metastases in the glands, the liver, the spleen, etc., is of frequent occurrence. The disease runs its course under the type of a chronic pulmonary affection, and almost always ends fatally. The endemic occurrence seems to point to an infectious origin for the tumor.

2. ECHINOCOCCUS OF THE LUNGS

Primary echinococcus of the lungs is very rare. In most cases the echinococci are brought to the lungs secondarily from other organs, either by way of the blood current, or, as is far oftener the case, by perforation of an echinococcus of the liver through the diaphragm.

The symptoms of echinococcus of the lungs are manifold. The parasite sometimes remains entirely concealed. In other cases a more or less severe, and often febrile, affection of the lungs, is developed, with pain in the chest, cough, and sometimes bloody expectoration, and dyspnœa. Physical examination of the lungs gives in some cases dullness, absence of respiratory murmur, and diminished vocal fremitus, while after the expectoration of the echinococcus symptoms of a cavity may ensue. A correct interpretation of all these symptoms is possible only when the echinococcus cysts are coughed up, or parts of them, like the membranes of the hooklets, are found in the expectoration. In some cases of echinococcus, the sputum is of a peculiar ocher-yellow color.

The termination of the disease may be favorable if the echinococci are expectorated, or if we succeed in removing them by operative means.[1] We can hardly hope to be able to kill the parasite by inhalations of turpentine or benzine. Sometimes the echinococcus cyst becomes gangrenous or suppurates. Rupture into the pleura, into the peritoneum, into the pericardium, and externally, has also been observed. This last termination is the most favorable, since otherwise, if the affection progresses, a fatal result may be caused by the sequelæ, or rarely by the occurrence of suffocation. The details of the natural history of the echinococcus are given under echinococcus of the liver.

[1] The recent introduction of the various Sauerbruch and Brauer air-pressure cabinets, and the Meltzer-Elsberg apparatus for direct inhalation of compressed air through an intratracheal tube, have revolutionized thoracic surgery. Brilliant results have already been reported from operations upon tumors, abscesses, and other conditions of the pleura, lungs, and mediastinum and upon diverticula, stenoses, and neoplasms of the œsophagus. With increased experience, improvement of apparatus and technic must follow, and if later results bear out the promise of these early successes, the prognosis of many conditions within the thorax will become more hopeful.

3. PULMONARY SYPHILIS

This would also be the place to speak of syphilitic new growths in the lungs, but, in our opinion, in spite of the quite abundant literature of this subject in recent times, no definite clinical description of pulmonary syphilis can be given. Those physicians who are disposed to consider every pulmonary disease in a previously syphilitic subject to be of a syphilitic nature, certainly regard many things as pulmonary syphilis which have nothing at all to do with syphilis. At least, we have found that all those cases which at first suggested a diagnosis of pulmonary syphilis, finally, upon more accurate examination and after longer observation, have turned out to be something else, usually tuberculosis. There are a few indubitable cases of chronic indurative contraction of the lungs following primary syphilitic infiltration. The clinical picture here does not differ in any characteristic manner from that of ordinary chronic interstitial pneumonia. A probable diagnosis may be made from the knowledge of the syphilitic taint, the simultaneous existence of other syphilitic lesions—e. g., of the liver—the physical signs of chronic pulmonary disease, and the failure to find tubercle bacilli in the sputum on repeated examinations. It is also well established that there may be syphilis of the larger and medium-sized bronchi, which is recognized at the autopsy by extensive radiating cicatrices in the bronchial mucous membrane, which sometimes lead to stenosis. Single gummatous nodules in the lungs are of the greatest rarity. We sometimes find in the pleura peculiar radiating cicatrices, which probably are of syphilitic origin. The pulmonary syphilis of the newborn, which occurs in the form of single nodules or as a diffuse syphilitic infiltration, the so-called *pneumonia alba,* has only a pathological interest.

If the existence of a syphilitic disease of the lungs is suspected, of course specific treatment should be employed. Iodid of potassium must be administered and perhaps also a course of mercurial inunctions. The results are not apt to be brilliant, however, because it is, of course, impossible to restore the portions which have been cicatrized.

SECTION V

Diseases of the Pleura

CHAPTER I

PLEURISY

(*Pleuritis*)

ÆTIOLOGY

Pleurisy is divided into primary and secondary. In a strict sense, the only cases of pleurisy which can be termed primary are those in which the pathogenic organisms penetrate into a healthy body, and become localized in the pleura without any previous organic lesion elsewhere. No doubt many a

case of pleurisy seems primary from a clinical standpoint, which is really not so. The disease antecedent to the pleurisy may be so slight as to present no symptoms and attract no attention. The pleurisy appears as an apparently independent disease, and is the first thing which attracts attention to the previously existing affection.

Exclusive of traumatic pleurisy, resulting from such injuries as a penetrating thoracic wound, we know positively of only one form of pleurisy—viz., the rheumatic—which is primary. This is, from an ætiological point of view, most closely allied with acute articular rheumatism (Fiedler). In polyarthritis (*q. v.*) it is not very exceptional to have pleurisy develop secondarily. In many cases the rheumatic affection attacks the pleura at first, without any great involvement of the joints. Then often follow articular trouble, endocarditis, and the like, confirming, or for the first time disclosing, the ætiology of the attack. Whether still other pathogenic influences may produce primary pleurisy is not settled. Some cases of acute pleurisy, setting in with high fever, we have felt justified in referring to diplococcus infection, as suggested by the association of herpes and other symptoms. In such instances it is scarcely possible to exclude with certainty the presence of a small focus of pneumonia. In genuine primary pleurisy the pathogenic germs must first enter the circulation, and by that means reach the pleura.

Among those varieties of pleurisy which appear from a clinical standpoint as primary, while their existence is really referable to a previous lesion of the body, by far the most frequent and important is the tuberculous. On this point it was only gradually and by means of long experience that physicians obtained a correct conception of the truth. We believe it right to say that the great majority of all cases of apparently primary pleurisy are tuberculous. The infection of the pleura is due to the invasion of it by germs from some tuberculous focus, situated near by. Either there are small tuberculous patches in the lungs which extend to the pleura, or, probably yet more frequently, bronchial or retrobronchial lymph-glands, being tuberculous, break into the pleural cavity and promptly excite pleurisy. In many of these cases the further course of this disease clearly shows that the pleurisy was tuberculous at the start, and not infrequently the physician is justified, even at the very beginning of the illness, in expressing his suspicion of tuberculosis.

In many other cases of pleurisy its secondary character is clear from the start. Most of these occur from the direct extension of the inflammatory process from some neighboring organ to the pleura. When considering pulmonary diseases, we had occasion to point out that the various pathological changes in the lungs, when they extend to the pleura, involve it in the disease. Thus we see pleurisy associated with croupous pneumonia, lobular catarrhal pneumonia, pulmonary gangrene, hemorrhagic infarction, embolic abscesses, and, most important of all, pulmonary tuberculosis. Many of the diseases just enumerated are frequent complications of the most diverse diseases. Hence, it is easy to understand that pleurisy is a not infrequent phenomenon in all sorts of severe illnesses.

Because of their practical importance, the rather frequent pleurisies of the puerperium and those following major operations should be particularly mentioned in this place. Puerperal pleurisy is sometimes tuberculous. We know that the post-partum period often favors the outbreak of a previously

latent tuberculosis. Very often the pleurisies are of an embolic nature, and depend on small emboli of the lungs (*vide supra,* page 325), whose source is to be sought for in thrombosed uterine, femoral, and other veins. Finally, pleurisy may be one of the manifestations of a general sepsis. Pleurisy following major operations is almost always of an embolic nature and results from thrombosis of veins following operative trauma.

Inflammation from other neighboring organs besides the lungs may extend to the pleura. Inflammation of the contiguous serous membranes may spread to the pleura by continuity; thus pleurisy complicates pericarditis and peritonitis. Inasmuch as the pleura and the outer surface of the pericardium lie in direct apposition and, furthermore, the pleural cavity and the peritoneal cavity are directly connected by the lymph-channels of the diaphragm, we can easily understand that not only serous and purulent, but also diplococcus, inflammation of the pericardium and peritoneum may have pleurisy for a sequel.

Another variety of secondary pleurisy is due to the conveyance of the inflammatory organisms not from the immediate neighborhood, but from other parts, by way of the circulation. Here should be mentioned pleurisy due to general sepsis, to polyarthritis, to nephritis (*q. v.*), to genuine gout (*q. v.*) and the like. The factors which give rise to the inflammation are either organic or, in the case of gout and nephritis, chemical in their nature. The inflammation they excite in the pleura is most varied in its kind and degree.

Of late years bacteriological investigations have been made by E. Levy, Prince Ludwig Ferdinand, and others, to obtain a more accurate knowledge as to the special variety of germs in the various forms of pleurisy, due to infection with organized material. It must not be forgotten, however, that these bacteriological investigations have been, for the most part, directed not to the diseased tissue, but to the inflammatory pleuritic exudation. This, however, in many cases contains no bacteria whatever. In particular, the exudation in most cases of tuberculous pleurisy, whether serous or purulent, is absolutely sterile, and in the secondary pleuritic exudations, in acute polyarthritis and nephritis, the search for bacteria has failed again and again. In other cases of pleurisy, however, staphylococci have been found in the exudate, and in empyema streptococci also. Metapneumonic pleurisy, whether serous or purulent, is often, though not always, characterized by the presence of genuine pneumococci. In general, the question with regard to the special cause of the disease will have to be whether the pleurisy is caused by the original primary agent of the disease (e. g., tubercle bacilli in tuberculosis, diplococci in pneumonia, etc.) or whether it is to be considered as a secondary complication (streptococci in typhoid fever, for example).

Special predisposing causes (above all, colds, and occasionally trauma) must be taken into account in some of the cases in all the different varieties of pleurisy. Positive judgment on these factors is often a very difficult matter.

PATHOLOGICAL ANATOMY

The inflamed pleura is markedly injected, it has lost its normal luster, and instead has a dull surface. This dullness is due to the coagulated fibrinous exudation upon the pleura, the exudation, in mild cases, forming only a thin layer. In more advanced cases, however, the surface of the pleura is covered

with thick, rough, and shaggy masses of fibrin. As long as the fluid in the pleura is little or not at all increased, we speak of a simple fibrinous or dry pleurisy (*pleuritis fibrinosa vel sicca*).

In other cases, however, besides the layer of fibrin there is an abundant exudation of fluid from the capillaries of the pleura, forming a pleuritic effusion. This is ordinarily of a simple serous character—serous and serofibrinous effusions. The fluid collects between the surfaces of the pleura, or, if there is at the same time an abundant coating of fibrin, between the gaps and in the meshes of the fibrinous exudation. In such cases there are often many flakes of fibrin floating in the fluid.

Upon microscopic examination there are invariably found a few leucocytes even in serous exudations (detailed description of the special varieties of leucocytes in the effusion is given in the section on the diagnosis of pleurisy), and also sometimes a very few blood corpuscles and endothelial cells (often swollen or fatty-degenerated), and plates of cholesterin.

If the number of pus corpuscles in the exudation becomes much increased, we have a seropurulent or a purulent exudation. This is always due to the presence of a specific organized poison which excites the suppuration. The pleurisies which come from embolic abscesses, from gangrenous foci in the lungs, and from carious ribs, and those which arise from the rupture of tuberculous cavities into the pleura, are almost always of a suppurative character. We call the purulent pleuritic effusion empyema. If putrefactive agencies enter the pleural cavity at the same time with the pus poison, as in the pleurisies which develop in pulmonary gangrene, the purulent exudation assumes an ichorous, putrid character—ichorous effusion.

Under certain circumstances the effusion assumes a hemorrhagic character —hemorrhagic effusion—especially if hemorrhages occur from the old or newly formed capillaries dilated by the inflammation. They arise partly by diapedesis and partly from rupture of the walls of the vessels. The exact cause of the hemorrhages is usually unknown. We know by experience that hemorrhagic effusions are most frequent in tuberculous pleurisy, a fact which is of diagnostic importance. The exudation may also be hemorrhagic in connection with new growths of the pleura, after severe croupous pneumonia, in septic disease (e. g., puerperal fever), and finally when there is a general hemorrhagic diathesis, as seen in scurvy, purpura hemorrhagica, and leukæmia. In all these last-mentioned cases it should, of course, be considered that we are not always dealing with a true inflammatory exudation, but merely with a hemorrhage into the pleural cavity.

The amount of fluid collected in one pleural cavity is, in the majority of cases, somewhere between a pint and a quart (500 to 1,000 c.c.), but it may reach three or four quarts. Every large effusion must influence the position of the yielding walls of the pleural cavity, the chest wall, the lungs, the mediastinum, and the diaphragm, through the consequent increase of pressure in the affected pleural cavity; and the resultant symptoms of pressure on the neighboring organs are of the greatest clinical significance. Attention is first called to the lungs themselves. Since the normal lung is expanded in the thorax beyond its elastic equilibrium, it will retract as soon as a part of the pleural cavity is occupied with fluid. Until it has reached its position of elastic equilibrium there can be no question of a positive pressure on the lung. The lung

floats on the effusion, in a certain way, if there be no adhesions, but, as the amount of the fluid further increases, compression of the lung follows. With a very large effusion the lung is pushed wholly up and back against the vertebral column, and is changed to an almost bloodless, airless, flat mass. It is, however, possible that the atelectasis of the lung is not caused exclusively by compression from without, but that, after the normal respiratory movements have ceased, a part of the air in the lung may be absorbed by the vessels, or even by the effusion.

We also see the results of the pressure exerted by the pleuritic effusion on the mediastinum and diaphragm, as well as on the lungs. Displacements of the heart arise from the lateral pressure on the mediastinum, which must take place if the pressure in the diseased pleural cavity is equal to that of the atmosphere, for a greater and positive pressure is unnecessary, since a negative pressure prevails on the healthy side. The downward pressure of the diaphragm, which usually affects both halves of it, although in unequal degree, makes itself manifest on the right by the low position of the liver, and on the left by the downward displacement of the stomach and large intestine (*vide infra*). It must be particularly noticed, however, that adhesions may prevent all the pressure displacements which we have mentioned, both of the lungs and of the neighboring organs.

As regards the further changes and terminations of the pleuritic processes, they depend upon the amount and character of the effusion. Favorable cases may result in complete recovery and absorption of the effusion. The fluid contents are taken up directly by the lymphatics of the pleura, and the solid constituents, the fibrin and the white blood corpuscles, are decomposed, dissolved, and absorbed.

In most of the severe cases, however, an extensive new growth of connective tissue and blood vessels takes place. The fluid exudate is often absorbed in very large part, but the pleura itself becomes thickened and converted into a dense inflammatory exudate. Extensive softer or firmer adhesions between the visceral and parietal pleura are a very common sequel (adhesive pleurisy). Between the adhesions there may still be isolated areas in which remains of the fluid exudate may be encapsulated ("encysted pleuritic exudate"). In cases of pleural inflammation of long duration, and, especially, in frequently recurring cases (particularly those following chronic pulmonary tuberculosis), the dry pleuritic exudate may finally acquire a thickness of from 0.4 to 0.8 inch (1 to 2 cm.). Deposits of calcium salts and new growths of bone tissue are sometimes to be found in old pleuritic exudates. Such diseased conditions, when associated with pronounced retraction, are sometimes termed "pleuritis deformans."

Whenever a case of extensive pleurisy, with an abundant fibrinous or fluid exudation, gets well, there is a marked cicatricial contraction of the pleura, in which the whole thoracic wall is involved. It requires months for the lungs and the thorax to regain their normal expansion, if they can ever do so.

That recovery from large pleuritic exudation is so often incomplete, is explained for the most part by the nature of the original disease. We often find that temporary improvement is followed by a fresh relapse of pleurisy, or by the appearance of extensive and usually tuberculous disease of the lungs or other organs.

In the case of purulent exudations, also, final absorption of the fluid is possible. This is especially likely when the empyema is metapneumonic and benign, but this demands much time, and thick, cheesy masses of pus are often left in the pleural sac. In most cases of empyema, if there is not timely operative interference, the pus seeks an outlet for itself. It may break through the visceral pleura into a bronchus, and be emptied externally, thus giving rise to a pyopneumothorax; but in many cases the pleura seems to be destroyed only superficially, and the pus is pressed into the alveoli as into a sponge, especially by the movements of coughing, and thence reaches the bronchi, without letting the air enter the pleural cavity (Traube). In other cases the empyema breaks externally through the chest wall—*empyema necessitatis*. The point of rupture is usually found in the vicinity of the sternum, where the chest wall is thinnest. In very rare cases the empyema breaks through the deeper parts of the trunk, or into the abdominal cavity.

Course of the Disease.—We will speak in what follows especially of the course and symptoms of ordinary, apparently primary (*vide supra*), fibrinous or serofibrinous pleurisy, the so-called simple pleuritic effusion. What is said of it obtains in large measure in the other form of pleurisy also. The physical signs, of course, are almost wholly independent of the character of the effusion. As far as the different forms of pleurisy differ clinically, we will mention their peculiarities below.

Only rarely is the onset of pleurisy quite acute and sudden, beginning with a rigor. In such cases we must guard against confusing it with croupous pneumonia. Cases of pleurisy of embolic origin (*vide supra*) usually have a sudden onset. As a rule, however, pleurisy begins slowly and gradually. The symptoms, which the patient himself feels, are in many cases to be referred directly to the disease of the pleura. One of the most constant is the pleuritic pain, the stitch in the side. A more or less severe pain comes on in the side at every deep breath, and hence upon any physical exertion; also upon movements of the body, in stooping, coughing, or gaping. Shortness of breath soon appears, and constantly increases. There is often a frequent, dry cough. Sometimes there are scarcely any cough and expectoration. Such expectoration as there may be is usually simply mucous. Besides that, severe general symptoms almost always develop; the patient feels dull, looks pale, and has no appetite. Patients who can endure a good deal often keep at work for a long time, until, after feeling miserable for three or four weeks, they are forced to stay at home and summon a physician. It is very important to know that in not a few cases the general symptoms are much more prominent, at the beginning of pleurisy, than the local ones. The patient comes to the physician complaining only of weakness, loss of appetite, or headache, and the physical examination is the first thing that shows the presence of perhaps a large pleuritic effusion.

In most of the severe cases the further course is slow like the beginning, but sometimes the severest symptoms, most intense dyspnœa, marked cyanosis, etc., may come on in a short time, owing to a sudden increase of the effusion. On the other hand, in mild cases the symptoms may disappear again in a few weeks, but the objective signs in such cases are generally to be made out for a longer time. The disease ordinarily lasts for at least five or six weeks, and

often much longer. Gradual recovery follows, or some new disease, usually tuberculous (*vide supra*).

SYMPTOMS

Single Symptoms.—*Pleuritic Pain.*—The pleuritic pain, the stitch in the side, is one of the most frequent subjective symptoms. We have previously mentioned that in primary diseases of the lungs, too, as in croupous pneumonia, the stitch in the side is due to the accompanying pleurisy. It is remarkable that the intensity of the pain in no way corresponds to the apparent intensity of the disease. There is often the severest pain in the side when the physical examination shows almost nothing. On the other hand, we often hear a decided pleuritic rub without the patient's complaining of any special pain. Pressure on the chest wall on the affected side is often very painful. With severe pain we may consider the possibility of an invasion of the intercostal nerves by the inflammation. We believe we have observed a few cases, like those described by some authors, of "crossed pleuritic pain"—that is, cases in which the pain is localized on the side not affected.

Cough and Expectoration.—The cough is probably excited directly by the disease of the pleura. We often find that the pain in the side and also the cough are brought on by a deep inspiration. Expectoration is entirely absent in uncomplicated pleurisy, or it is scanty, and consists simply of mucus. Much expectoration always means a pulmonary complication. A large amount of purulent sputum is evacuated if a purulent effusion breaks into the lungs (*vide supra*).

Dyspnœa.—The respiration is usually shallow, and consequently frequent, because of the pleuritic pain. In every large effusion which prevents respiration in one lung the dyspnœa becomes more severe, and may, with a very extensive effusion, reach the highest degree of orthopnœa. The stronger the patient was before the disease, and the more rapidly the effusion develops, the more severe, as a rule, is the dyspnœa.

Fever.—Most severe pleurisies are associated with fever, but its height is not very great, so that it quite rarely reaches 104° F. (40° C.). The fever has no typical course. In cases with an acute beginning it is sometimes quite continuous, or slightly remitting at first. If improvement takes place, the fever goes down in about two or three weeks by lysis, so that this part of the temperature curve may be precisely like the period of defervescence in typhoid.

In the more protracted cases the fever gradually becomes more remitting, varying between 100° and 101° F. (38 to 38.5° C.), and it assumes more and more the form of hectic fever. The longer the evening rise of temperature lasts, the more we are justified in suspecting tuberculosis. In empyema we see a higher, irregular fever, sometimes associated with severe chills.

The pulse is constantly rapid, up to 100 and over. In all severe cases the strength and tension of the pulse are much diminished. Irregularity of the pulse is not infrequent. All these changes are probably due in great part to the pressure of the effusion on the heart and large vessels. Lichtheim has discovered experimentally that it is not the compression of the vessels in the compressed lung which lowers the arterial tension.

General Symptoms.—Pleurisy is almost always associated with a pronounced general malaise, muscular weakness, and dullness. The patient is

pale, and often markedly cyanotic in cases with much disturbance of respiration. There is great emaciation if the disease is of long duration.

The appetite declines from the outset. There is often occasional vomiting, especially in the first period of the disease. The bowels are usually constipated. Many patients complain of headache.

The condition of the urinary secretion is very important. In every pleuritic effusion the amount of urine is decidedly diminished so long as the effusion increases or remains at the same height. The daily amount is sometimes only 8 to 13 ounces (200 to 400 c.c.). The urine is also concentrated, its specific gravity being about 1.020 to 1.028. Sediments of urates often form. This diminution of the excretion of water by the kidneys is generally referred to the diminished arterial pressure.

But other unknown conditions (the retention of sodium chlorid, possibly) also play a rôle. Even in cases of only moderate exudation it is remarkable how long the urinary excretion is diminished before it finally becomes normal. Therefore, an increase of the amount of urine is always a favorable symptom, often the first sign of beginning absorption of the effusion. If a large effusion is rapidly absorbed, the amount of urine may increase to 80 or 100 ounces (2,500 to 3,000 c.c.) daily. The urine, then, of course, is abnormally clear and of low specific gravity. This pronounced diuresis often persists for so long a time during the period of absorption of pleural exudates that here, also, other factors than the mere elimination of the absorbed fluid must be taken into account.

Physical Signs.—1. Fibrinous Pleurisy — Pleuritis Sicca.—Simple fibrinous pleurisy sometimes gives rise to no physical signs at all. If it develops as a result of some pulmonary affection, the physical signs present are often dependent upon the latter alone.

In many cases, however, dry pleurisy may cause marked objective signs. On inspection, we are struck by the impaired mobility of the affected side on respiration, which is due to the pain caused by breathing. Because of this same tenderness the patient at first often lies on the sound side. Percussion gives no qualitative change of resonance as yet. With the beginning of exudation slight dullness appears, at first almost always in the posterior portion of the lungs. Sometimes the resonance becomes tympanitic as a result of retraction of the lung. We can almost constantly make out, especially in the back, that the lower edge of the lung moves less than usual on respiration. Auscultation gives a respiratory murmur that is either qualitatively unchanged or indefinite, but it is always diminished. The peculiar pathognomonic sign of dry pleurisy, however, is the pleuritic friction rub, that characteristic rubbing, grating, creaking sound which arises from the sliding of the rough pleural surfaces over each other, and is detected especially in the lateral portions of the thorax. We can hear it both on inspiration and on expiration. It is often jerky, one rub following another after a considerable interval. If we are sure we hear a pleuritic rub, it is direct evidence of the existence of a dry pleurisy, but its absence will not let us exclude pleurisy. The friction sound must be absent if there are pleuritic adhesions. We can often feel a marked rub by laying the hand on the chest. Sometimes the patient feels it himself, but in other cases he has no sensation of it. We may confound a slight rub with fine crepitant râles. Repeated examinations before and after the

patient has coughed usually confirm the diagnosis, since the râles are often changed by coughing.

To be distinguished from the mild form of dry pleurisy just described is that variety in which there is an extensive and abundant fibrinous exudation, with scarcely any liquid. We have repeatedly seen cases following pneumonia, polyarthritis, or even apparently primary cases, which displayed marked dullness and sense of resistance on percussion over the whole of one side of the chest, with diminished or absent respiration, and yet upon exploratory puncture only a few drops of serous exudation could be obtained. Evidently we had to do with the formation of large amounts of coagulated fibrin. Attacks of this sort usually run a severe and tedious course, but they may, nevertheless, get well at last.

2. Pleuritic Effusion.—Small amounts of fluid in a pleural cavity escape discovery. Physical signs first appear when the amount of effusion reaches 8 to 10 ounces (200 to 300 c.c.).

Inspection shows first the more or less marked impairment of motion on the affected side on respiration. If the amount of the effusion is large, there is an evident distention of the affected side in the lower posterior and lateral portions of the thorax. The intercostal spaces are flattened or even a little convex. The nipples and shoulder blades are farther removed from the median line on the affected side than on the healthy side. The hypochondrium on the affected side is more prominent. In an extraordinarily large effusion on the left side we have seen and felt, in the left hypochondrium, the lower surface of the diaphragm, which was actually arched downward. By direct measurement in severe cases we can make out that the affected side is expanded several centimeters.

With every large effusion there is marked dyspnœa and accelerated respiration. The slight excursions of the affected side on respiration are usually very striking, while the sound side moves so much the more. In this stage of pleurisy the patient often lies upon the affected side, in order to breathe with the healthy lung with as little restraint as possible. With large effusions complete orthopnœa may develop.

The signs due to displacement of the neighboring organs, which are noticeable on inspection, will be mentioned below in the appropriate connection.

Everywhere that a layer of fluid comes between the lung and the chest wall there is a loss of clearness in the percussion note. If the thickness of the layer of effusion is 5 or 6 cm., the resonance seems completely dull or flat. The pleuritic dullness is almost always made out first in the lower posterior portions of the thorax, more rarely in the lower lateral portions. With a slight effusion the height of the dullness is only a few centimeters, but, with much effusion, it rises higher in the back and the lateral portions of the thorax; and on the right, resonance gradually grows dull anteriorly and inferiorly, above the liver. With very large effusions the dullness may begin in front at the second or third rib, or in rare cases even the whole half of the chest, front and back, may give a totally flat percussion note. Pleuritic dullness is always attended with a marked feeling of resistance on percussion.

With medium-sized effusions, when the dullness does not extend over the whole back, the upper boundary of the dullness usually forms an oblique line, highest at the vertebral column and thence running obliquely downward to the

side of the thorax. This is especially observed in cases that have taken to their beds early in the disease; under such circumstances the exudate must naturally collect in the posterior portions of the pleural cavity. The upper border of the dullness has a more horizontal line in patients who, despite beginning pleuritic effusion, are up and about a good deal. Again, the highest level of the upper border of dullness is not infrequently found in the posterior axillary line; from this point the line of dullness drops somewhat, both anteriorly as well as toward the vertebral column. This depends, in part, on the lateral posture so often assumed by the patient (*vide supra*), and, in part, on the compression of the lung against the vertebral column. Hence a more tympanitic percussion zone may develop posteriorly and below alongside the vertebral column. Occasionally, when there is very large effusion into the left pleura, a small area of dullness may also be found on the normal side, posteriorly and below, near the vertebral column (Rauchfuss, Grocco). It is probably principally dependent upon the displacement of the mediastinum. An alteration in the position of the patient (lying down or sitting up) generally causes very little or no change in the upper border of the dullness, because of the permanent or temporary union of the pleural surfaces. On the right it is not possible to distinguish by percussion the lower limit of the exudation from the liver. On the left we can often distinguish its lower limit from the tympanitic resonance of the stomach, and are thus aided in diagnosis (*vide infra,* displacement of organs).

The percussion note above a pleuritic effusion deserves attention. The beginning of pleuritic dullness is almost always relative, gradually passing to an absolute flatness. The pulmonary resonance above the beginning of dullness is usually tympanitic, from retraction of the lung tissue. We find the tympanitic resonance beautifully distinct in large effusions in the first and second intercostal spaces in front. It is loud and deep, and remains unchanged with the mouth open—Skoda's resonance. With very large effusions, which cause an actual compression of the lung, we sometimes find, in the second intercostal space, a dull tympanitic resonance, which becomes higher on opening the mouth. This resonance arises from the vibrations of air in a large bronchus surrounded by compressed lung—"Williams's tracheal tone." With large effusions we sometimes hear over the retracted lung, in the upper anterior intercostal spaces, a distinct buckram sound—the "cracked-pot sound."

Displacement of the neighboring organs, which is made out chiefly by percussion, forms one of the most important physical signs in pleurisy with effusion.

In right-sided effusions the liver, especially the right lobe, is displaced downward. We find the lower border of the liver dullness extending several centimeters below the ribs. In very large effusions the liver may be pushed down to the level of the umbilicus. The pushing of the mediastinum to the left in large effusions may be recognized upon percussing from the right toward the left, by dullness over the upper part of the sternum, reaching to or beyond the left border of the sternum. The displacement of the heart to the left in the majority of well-marked cases is associated with a displacement of the apex of the heart upward. This is easily explained by considering the position of the heart and the direction of the pressure, which first acts from below. We recognize the displacement of the heart chiefly by the position of the apex

beat, which is seen and felt at or outside the left mammillary line in the fifth space, or often higher, as we have said—in the fourth. Percussion gives a corresponding displacement of the left boundary of the cardiac dullness to the left.

In left-sided effusions the displacement of the heart to the right, which can usually be made out even in moderate effusions, is especially noticeable. Resonance over the lower part of the sternum is diminished, the heart's dullness extends to the right border of the sternum or several centimeters beyond it. In the most marked cases the heart is pushed to the right mammillary line. The displacement of the mediastinum is also to be made out over the upper part of the sternum, the dullness reaching to the right border of the sternum or beyond. The low position of the diaphragm is made out by a depression of the left, and in marked cases of the right, lobe of the liver. It is an especially important sign, however, that dullness occurs in the zone, about a hand-breadth wide, of normal tympanitic resonance above the left border of the ribs —the "semilunar space" of Traube. The normal tympanitic resonance here comes from the stomach or large intestine. As the diaphragm is pressed downward the pleuritic effusion presses on these organs. The semilunar space is therefore diminished, and finally, with large effusions, there is absolute dullness down to the edge of the ribs.

Changes in dullness in pleuritic effusions may occur with a change of the patient's position, but they may often be absent on account of adhesions. As a rule, the normal change in the position of the lower border of the lung corresponding with respiration is abolished.

Auscultation always gives a diminished respiratory murmur over the pleuritic effusion. With a beginning effusion it may sound approximately vesicular, but later it becomes indefinite, harsh, and finally bronchial, if the larger bronchi remain open for the respiratory current of air. The bronchial respiration sounds distant and low, and has the character of the sharp German *ch*, but in rare cases it also assumes a distinct amphoric tone, so that it sounds almost like a cavernous respiration. The respiratory murmur may finally disappear entirely over very large effusions. Above the upper boundary of the effusion the respiration almost always sounds harsh. Among the adventitious sounds we must mention the pleuritic friction sound, which of course can be heard only at the upper boundary of the effusion, where the two pleural surfaces meet. Moist râles and rhonchi signify a coexisting disease in the lungs. With slight effusions we often hear, on deep breathing, pure crepitant râles on inspiration, as the walls of the alveoli and bronchioles in the atelectatic lung, which were stuck together, are torn apart.

On auscultation of the voice we sometimes hear bronchophony, and sometimes that bleating, nasal sound known as ægophony. Baccelli advanced the theory that auscultation of the whispered voice might be of service in diagnosticating the character of the effusion. With a serous effusion we can understand a whisper distinctly through the thorax, but not with a purulent effusion, since theoretically the cell elements destroy the waves of resonance. This theory holds true in many cases, but by no means in all.

On auscultation of the heart we notice, as a result of its displacement, an abnormal etxension of the region over which the heart sounds are audible. If the inflammation spreads from the pleura to the outer surface of the peri-

cardium, we can sometimes hear an extrapericardial friction rub, accompanying both the respiration and the action of the heart.

The vocal fremitus is always diminished over the pleuritic effusion, and in marked cases is entirely absent.

3. Absorption of the Effusion—Pleuritic Contraction.—The beginning absorption of the effusion is usually first made evident by the percussion note in the upper part of the dullness becoming clearer and sometimes tympanitic. The respiratory murmur is also plainer. Where it was bronchial it becomes indefinite and gradually vesicular again. The vocal fremitus is again to be felt. All these improvements take place gradually but slowly. It is usually a very long time before the percussion note resumes its normal clearness.

The changes in the form of the thorax are especially striking. Only in pleurisies with slight effusion does the somewhat expanded thorax resume its old form without further change. After every severe pleurisy with large effusion there is, during its absorption, a marked contraction of the affected half of the chest. In cases of moderate intensity the contraction principally affects only the lower lateral portions of the thorax, in severe cases the upper and anterior portions as well. We find the most marked contractions in children and young persons with a yielding thorax. The circumference of the affected side is much less than that of the sound side. The ribs are pressed together and the intercostal spaces become very narrow. The fossæ are deepened and the nipples and shoulder blades are drawn nearer the vertebral column, which takes on an abnormal lateral curvature, in which its convexity is directed usually toward the affected side, but sometimes to the sound side. Dullness and diminution of the respiratory murmur and vocal fremitus continue with the contraction of the pleura, but they no longer depend upon the presence of a fluid effusion, but are due to the pleuritic thickening.

The process of marked contraction always lasts for months, or even longer. In favorable cases the contraction of the thorax may be readjusted very much later, often after years. The thickening is absorbed, and the lungs and thorax gradually expand, but in other cases there are extensive adhesions between the pleural surfaces, especially over the lower lobe, which result in a permanent disturbance of respiration. In almost all cases of pleurisy with contractions there arises a vicarious emphysema in the lung on the sound side.

COMPLICATIONS

Peculiar complications of pleurisy are rare. Where such occur they are due either to the primary disease which has led to the pleurisy, or to the simultaneous action of the same cause of disease, such as tuberculosis. Hence it happens that we speak of the frequent "complication" of pleurisy with chronic bronchitis or with tuberculosis of the lungs or other organs. It is important to bear in mind that, by a direct advance of the inflammation, the pleurisy may also invade the pericardium, and rarely the peritoneum, through the diaphragm; but we see this extension of the process almost solely in tuberculous and purulent pleurisies. We must mention, finally, that we have seen several cases with a large serous effusion, in which an acute hemorrhagic nephritis occurred. For the paralysis of the arm on the correspond-

ing side observed in some cases of empyema, compare what is said in regard to reflex paralyses in Volume II.

VARIOUS FORMS OF PLEURISY

1. **Primary Rheumatic Pleurisy.**—As we have already pointed out, when considering the ætiology of pleurisy, clinical observations have lately led to the belief that some of the cases of acute primary pleurisy are due to the same cause as acute articular rheumatism (which see). Although there are as yet no sufficient number of decisive bacteriological investigations, we are, nevertheless, strongly inclined, from our present experience, to acknowledge the correctness of this view, even if we do not estimate the frequency of rheumatic pleurisy nearly so high, as, e. g., does Fiedler, on the ground of his observations in Dresden.

Rheumatic pleurisy usually develops rather suddenly in persons previously healthy. Sometimes it is preceded by mild rheumatic pains in the muscles or some of the joints. The local discomforts, such as pain in the side, are often very severe, the fever is moderately high, though rarely over 104° F. (40° C.); and as the case progresses such rheumatic symptoms as swelling of the joints and endocarditis may also associate themselves with the pleurisy and confirm the diagnosis. In general, however, the course of the disease is favorable. The fever lasts, as a rule, only a week or two, and even when there is a large exudation it finally undergoes complete absorption with recovery.

2. **Tuberculous Pleurisy.**—In an ætiological sense we must declare by far the larger part of the ordinary "pleuritic effusions," which clinically seem primary, to be tuberculous. The further course of the cases, if we can watch them long enough, almost always permits us finally to recognize the tuberculous nature of the disease; yet we cannot say that some other tuberculous disease, particularly phthisis, is always the immediate sequel of the pleurisy.

In a comparatively small number of cases do the symptoms of acute tuberculosis, or more frequently of chronic phthisis, appear as an immediate result of the pleurisy, which at that time is usually still present or in the contracting stage. The objective changes of phthisis are evident either in the apex or in the lower lobe of the affected side. The fever continues, the pulmonary affection advances, the other lung is also attacked, and the disease takes a fatal course under the type of an ordinary phthisis, now more acute and now more chronic.

In other cases acute tuberculous affections arise sooner or later as a result of the pleurisy—tuberculous meningitis or general miliary tuberculosis. In other cases still the disease develops under the form of tuberculosis of the serous membranes, to which we will return again in the description of tuberculous pericarditis and tuberculous peritonitis. We often have to do with a double pleurisy with no evident complication in the lungs. In varying succession are added the symptoms of chronic tuberculous peritonitis, with pain, swelling, and effusion of fluid into the abdomen, or the symptoms of tuberculous pericarditis. Death finally ensues with persistent hectic fever and increasing general emaciation and weakness. The whole affection usually runs a chronic course, lasts for months, and often shows marked remissions and temporary improvements.

In very many instances, on the other hand, the pleuritic effusion has throughout an apparently favorable course. After some weeks the fever ceases, the effusion is absorbed, the patient gets up, and is finally discharged as nearly well. Of course, some dullness and retarded motion often remain in the affected side, but even these may gradually disappear. These cases, too, very often turn out in the end to be tuberculous. After a longer or shorter period of apparent health, sometimes after the lapse of years, a "new" disease appears—that is, either a return of the pleurisy, a pleurisy on the other side, or some other acute or chronic tuberculous affection. In such cases, too, we must look upon the former pleurisy, in an ætiological sense, as tuberculous. It is not impossible, however, for even a tuberculous pleurisy to recover, and for the recovery to be permanent, if no other organ is at the same time affected by tuberculosis, especially if the lungs remain intact.

Finally, we must mention the cases in which a pleuritic effusion develops secondarily to an already pronounced phthisis. Here, too, we almost always have to do with a tuberculous pleurisy.

The anatomical changes in tuberculous pleurisy consist in the ordinary signs of inflammation, and also the presence of the specific nodules of tubercle. The number of tubercles differs very much in different cases. The pleura is in some cases completely studded with miliary nodules, and in others we find the tubercles, at least with the naked eye, only in single spots. The effusion is usually of a serofibrinous character. Sometimes it is hemorrhagic, as the majority of cases of apparently primary "hemorrhagic pleurisy" are generally of a tuberculous nature. Empyema also (*vide infra*) occurs not infrequently in association with tuberculosis; and finally, in a few cases the exudation has been of a peculiar milky character, and has contained numerous oil globules, probably originating from fatty-degenerated and disintegrated leucocytes and endothelium.

3. **Purulent Pleurisy—Empyema.**—A purulent exudation is developed in the pleura when the inflammation of that organ is due to a specific cause which excites suppuration. So far as is yet known, the *Streptococcus pyogenes* seems to be the most frequent factor in producing empyema. It is found in the pus of empyema, due to such causes as external injury, caries of the ribs, pulmonary tuberculosis, pneumonia, and pyæmia. Less often empyema is excited by staphylococci. This is almost always a rather favorable form of the disease. Likewise comparatively benign is metapneumonic empyema, which in most instances is caused by the pneumococcus. The empyema of tuberculosis is, as has been already pointed out, in most cases devoid of bacteria (A. Fränkel and others), and here we may have to do with the formation of chemical poisons capable of exciting suppuration.

Empyema usually causes severe symptoms. The fever is higher than in the other forms of pleurisy, but is irregularly intermittent, and is often associated with chills. There are severe general symptoms besides the fever, such as great languor, headache, a dry tongue, and a rapid pulse. We sometimes notice a slight œdema of the chest wall on the affected side. Otherwise the local symptoms and disturbances are, of course, the same as in the other forms of pleurisy. If the pus is not evacuated artificially, the empyema may finally break externally or into the lungs (*vide supra*). In the latter case a very large expectoration of pus suddenly occurs, and is usually followed by pneumothorax.

DIAGNOSIS

Our chief attention in regard to diagnosis is directed to the distinction between pleurisy and acute or chronic pneumonia, which is not very easy in all cases. We will briefly contrast the distinctive features as made out on physical examination.

Inspection.—A marked distention of the affected side points to effusion; it does not occur in pneumonia.

Percussion.—The dullness in pleurisy is complete, and the feeling of resistance on percussion is very marked; in pneumonia, however, the dullness is rarely so marked, and there is often a tympanitic sound. The discovery by percussion of signs of displacement of the neighboring organs is of especial weight, as these signs are always absent in uncomplicated pneumonia, while with few exceptions they can be easily demonstrated in every case of pleurisy when the exudation is at all considerable.

Auscultation.—Diminished or suppressed respiratory murmur points to pleurisy, loud bronchial breathing and râles to pneumonia; but we must not forget that in pneumonia auscultation may give the same signs as in pleurisy, if a bronchus is plugged.

Vocal Fremitus.—Marked vocal fremitus over dullness is direct evidence of pneumonia, diminished or absent vocal fremitus of pleurisy; but the vocal fremitus may also be diminished in pneumonia if a bronchus is plugged.

Besides the physical signs, other phenomena to be observed are the mode of commencement, the course of the disease, the fever, the expectoration, and the appearance of herpes. The most reliable means of deciding in all doubtful cases is exploratory puncture, although even then we may, of course, be left in doubt whether an infiltration of the corresponding portion of the lung may not exist in addition to pleurisy. If the exudation is largely fibrinous, or if there is an abundant new growth of inflammatory connective tissue, the results of aspiration may be negative. Hence, in doubtful cases we should always make repeated trials.

If we have diagnosticated a pleuritic effusion, the next question is always as to the character of the effusion, because the prognosis and treatment are to a large degree dependent upon this. Although certain well-known ætiological circumstances, and the severity of the fever and the general symptoms, often permit us to suspect the nature of the effusion, whether serous or purulent, the only certain information comes from an exploratory puncture with a hypodermic syringe. If the syringe is carefully disinfected, and the operation is cautiously conducted, this procedure is entirely devoid of danger, and we must urgently advise its employment in all doubtful cases in order to settle the diagnosis. It is indeed of practical importance to know that there is a likelihood in empyema of obtaining no fluid by means of exploratory puncture (*vide supra*), even when, upon incision, pus is found. We have had this experience repeatedly. In doubtful and severe cases in which there is suspicion of empyema, it is therefore decidedly advisable, when life is threatened, to make an incision of the pleura. Besides a macroscopic inspection, a careful microscopic examination of the fluid withdrawn is sometimes of importance. Besides the ordinary constituents—red and white blood corpuscles, endothelial

cells, and cholesterin crystals—we may sometimes find something of special diagnostic significance, such as bacteria in septic pleurisy, carcinoma cells in cancerous pleurisy, etc.

We cannot always judge from the beginning whether a pleurisy is tuberculous or not; but we should never forget, as has already been affirmed, that in every case of pleurisy, even if it is apparently primary, there is a strong suspicion of tuberculosis. We must observe in particular the general habit and the nutrition of the patient, and inquire into the hereditary predisposition and any previous illness. In the further course of the disease persistent hectic fever, slowly increasing emaciation and pallor, fresh relapses, and the onset of pulmonary symptoms, point to the tuberculous character of the pleurisy. Every case of double pleurisy, and of pleurisy associated with pericardial symptoms, leads us most decidedly to suspect tuberculosis. Since tuberculous nodules of the serosa almost never ulcerate, tubercle bacilli cannot usually be found in those cases of tuberculous pleurisy in which microscopical examination of the centrifuged sediment is made. On the other hand, the tuberculous nature of the disease can often be demonstrated by the injection of about 10 c.c. of the fluid into the peritoneal cavity of a guinea pig; within four to six weeks tuberculous peritonitis develops. The examination of the sputum, too, is important, even in cases of apparently simple pleurisy. We have often been able to demonstrate tubercle bacilli in the scanty expectoration of such patients, even when there were scarcely any pulmonary symptoms. Careful microscopical examination of the cellular elements of the centrifugalized sediment of the aspirated pleural fluid (the "cyto-diagnosis" of Widal and others) is of considerable importance in determining the nature of the disease. We are nearly always dealing with a tuberculous pleurisy if mononuclear leucocytes compose the main part of the leucocytes present in the fluid. In cases of pleurisy from other causes, the cellular elements of the fluid are in largest part made up by the usual polynuclear leucocytes. As we have indicated, a hemorrhagic effusion is strongly in favor of tuberculosis, but also occurs in new growths of the pleura (*vide infra*).

The differentiation between inflammatory pleuritic exudates and pleuritic transudates (hydrothorax, *vide infra*) usually is evident from the general clinical picture of the disease, but it may occasionally be difficult. The percentage of albumen in the fluid should be estimated, it being much higher in exudates (usually not less than 4.5 per cent) than in transudates. Correspondingly, the specific gravity of exudates is higher (more than 1.015) than that of transudates. A considerable admixture of leucocytes in the fluid invariably speaks for its inflammatory origin. Endothelial cells alone are also present in considerable quantities in the transudation fluids as well as in exudates. Rivalta has devised an interesting test for the differentiation between exudates and transudates: A very weak solution of acetic acid (2 drops of glacial acetic acid to 3.3 ounces [100 c.c.] of water) is placed in a cylindrical vessel. A few drops of the aspirated fluid are dropped into this solution. In the case of an exudation, a readily visible, light cloud resembling "cigarette smoke" will appear in the path of the descending drops. This cloud is due to the precipitation of an albuminous body, and is entirely absent when a transudation fluid is tested.

PROGNOSIS

The prognosis, as regards the immediate danger of the disease, depends entirely upon the severity of the symptoms, and especially upon the dyspnœa. The prognosis, as regards the further course of the disease, depends chiefly upon the nature of the pleurisy. Many secondary and also many apparently primary pleurisies, although extensive, recover completely and permanently after weeks or months. Undoubtedly, an apparently complete cure not infrequently occurs also in cases of tuberculous pleurisy. A certain amount of apprehension for the future will, however, always be entertained, inasmuch as pulmonary tuberculosis only too frequently develops at some later date. The prognosis of empyema depends partly upon the underlying disease, but especially upon judicious and timely operative interference. In benign cases of empyema recovery is usually complete in a few weeks after operation, but sometimes months are required. The possibilities of a spontaneous rupture of empyema, internally or externally, have been mentioned above. With incomplete healing, which leaves a pleural fistula, we must fear the appearance of general amyloid disease in various organs.

In rare instances with large effusions sudden death occurs, an event which cannot always be satisfactorily explained. Probably there are different factors in different cases, such as pulmonary embolism, cerebral embolism, sudden cerebral anæmia, weakness of the heart, or the onset of pulmonary œdema.

TREATMENT

In the beginning of the disease the treatment is purely symptomatic. We try to alleviate the patient's symptoms, the pain and dyspnœa, by cold or warm applications (the latter are usually more grateful), sometimes, too, by dry cups, also by embrocations with chloroform liniment and sinapisms, with severe symptoms, by morphin internally or subcutaneously. Unfortunately, we have but few remedies to check the inflammatory process in the pleura. If an ice bag is well borne, it may be of service. The efficacy of the much-used painting with iodin is doubtful, but it may always be tried if there is a severe pleuritic pain. Perhaps more efficient is the application of iodized vasogen. In recent years the author has extensively employed soap inunctions with apparently very good results. Sometimes tincture of green soap is used, and sometimes a salve made of green soap, 20 parts, and 10 parts each of lanolin and vaselin. The inunctions are given once daily, care being taken to avoid any inflammatory eczema. If the skin is very sensitive, the inunction should be washed off with lukewarm water in an hour.

If a large serous effusion has formed, it is the universal custom to prescribe diuretics. The idea is to cause, or at any rate promote, the absorption of the exudation by increasing the excretion of urine. It is, however, very difficult to obtain any real proof of the usefulness of diuretics in pleurisy with effusion. They often are of no use whatever, but, again, the absorption of the fluid may follow the administration of a diuretic, so that it does not seem impossible that the latter has a therapeutic influence. With regard to a choice of the diuretics to be employed we would name, first, salicylate of sodium, 75 to 120 gr. (gm. 5 to 8) a day, because a specific influence has been ascribed to this drug in

pleurisy. This applies particularly to cases of supposed rheumatic origin (*vide supra*). Many good observers assert that primary pleuritic exudations often run a remarkably rapid and favorable course when treated with salicylate of sodium. Moreover, the drug has a direct diuretic effect. Besides this, the acetates of potassium and of sodium are frequently prescribed, as well as squills, and, of late, diuretin (sodio-salicylate of theobromin), given in doses of 15 to 30 gr. (gm. 1 to 2) two or three times a day. We have sometimes found this remedy apparently very efficient. If there are signs of cardiac weakness and diminished arterial tension, digitalis must be prescribed, alone or with a diuretic. Other internal remedies are at present seldom employed in pleurisy with effusion. The influence of iodid of potassium as an "absorbent" is extremely doubtful. Drastic purgatives and such diaphoretic remedies as hot packs and pilocarpin usually affect the general condition unfavorably, and are of little use. Probably there are to-day few advocates of the so-called Schroth method of treatment, according to which the amount of fluid ingested is reduced to a minimum, so as to promote the absorption of the exudation.

In many cases the evacuation of the effusion by puncture (introduced by Trousseau) is of the greatest importance. Many cases of pleurisy with effusion run a favorable course without it, and we consider it at least superfluous to puncture every effusion without sufficient grounds, but puncture is often one of the most serviceable therapeutic influences at our command, and may occasionally be a life-saving procedure. The first and most important indication for puncture is present when the effusion becomes so large as to be directly dangerous to life. As soon as the patient's dyspnœa reaches a dangerous degree, and the cyanosis becomes marked and the pulse weaker, a puncture must be made as a direct vital indication. When the exudation is of considerable size there may be a very sudden aggravation of the symptoms, so that in such cases one should not wait too long. It is much better to puncture too early than too late! Trousseau urged that tapping should invariably be performed when the dullness caused by the exudation involves not only the back, but also the whole or nearly all of the anterior wall of the thorax, a rule which the author almost always follows. The benefit of such a puncture is often pronounced. The second indication is a too protracted absorption of the effusion. Puncture is indicated if the effusion does not disappear after an apparent remission of the inflammatory symptoms, especially after the fever has gone. We often see the further absorption stimulated by such a puncture. It has been said that, if possible, tapping should be delayed until the fever has ceased, but to us this does not seem at all necessary. When the exudation was large, or the absorption was delayed, we have repeatedly aspirated even when the patient was still feverish, and have not infrequently found that the fever diminished remarkably upon the removal of the exudation (see the temperature chart, Fig. 58).

As regards the performance of the puncture, we cannot here go into all the numerous methods and forms of apparatus proposed. The distinctions are immaterial. The simpler the method, the easier it is to perform, and hence the better it is.

Every puncture must be preceded by an exploratory puncture in order to settle the diagnosis as to the presence and character of the exudation. A

medium-sized trocar with a lateral opening, to which a rubber catheter can be fastened, serves to evacuate the fluid. Billroth's and Fräntzel's trocars are useful. Aspiration can be performed still more conveniently with a hollow needle, and, best of all, according to our experience, with that proposed by Fiedler, which is now exclusively employed in the author's wards, because the point of this needle is guarded so that it cannot scratch the tissues, and also, if it becomes plugged with clots of fibrin these can be pushed out.[1] The

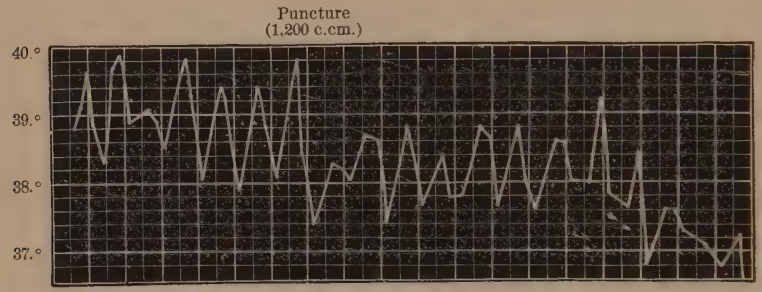

FIG. 58.—Temperature curve in pleurisy with effusion before and after tapping. (Erlangen Medical Clinic.)

instruments and the chest wall at the point of puncture must be carefully disinfected. We usually choose a point for puncture somewhere about the sixth intercostal space, in the middle or posterior axillary line. The patient sits up in bed, but is held and supported by another person, when it is possible. Before and during the puncture he takes a little strong wine, strophanthus, or similar stimulant. The pain of the operation may be mitigated by previously injecting morphin, or, still better, by causing local anæsthesia at the site of puncture with ethyl chlorid. In the overwhelming majority of cases, and especially if the exudation is abundant, we can remove a large part of the fluid by simple puncture with siphonage. With few exceptions, the pleuritic exudation is under a positive pressure of 10 to 25 mm. of mercury. The evacuating tube of the trocar must be previously filled with sterile water and conducted under a layer of the same into the vessel prepared to receive the effusion. The evacuation of the effusion should always be slow and gradual. Many physicians advise stopping when 1,500 c.c. have been removed, but if the fluid is allowed to escape slowly and everything is going well, this quantity may often be exceeded with impunity in the case of large exudations. While, as we have said, in most instances the exudation may be satisfactorily removed by simple puncture and siphonage, it will sometimes prove necessary to employ aspiration. Hence some physicians invariably do so, and to this there is no objection, even if it is unnecessary. The forms of apparatus most used for this are those invented by Dieulafoy and Potain. In puncture with aspiration we proceed more slowly and cautiously.

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[1] [Fiedler's instrument consists essentially of a sharp hollow needle, with an outer jacket, which can be moved so as to cover the sharp point after the chest wall has been penetrated. If the lumen is stopped up, a pointed steel wire thrust directly through the attached rubber tubing displaces the obstruction. Probably the least pain in tapping is felt after previous infiltration of the local tissues with 0.5-per-cent solution of cocain.]

[The necessity for two punctures—one exploratory, the other distinctly operative—does not seem clear. The two can be perfectly combined, a fair-sized trocar or needle being as easy of introduction and producing really no more pain than a very fine one, and being more sure to give results on which reliance can be placed. An ordinary Davidson's syringe makes a very satisfactory pump and can always be obtained.]

Unpleasant incidents which may cause a cessation of the process are rare. If the patient complains of dizziness or faintness we must cease, or, at any rate, pause. Usually everything goes well until the needle begins to rub against the pleura: then there is pain and generally a violent cough. It is well, then, to remove the needle at once. The cough can usually be promptly quieted by rest and a little morphin, but sometimes after aspiration there will develop a sort of pulmonary œdema, with the expectoration of a large amount of frothy serous sputum (*expectoration albumineuse*). This is perhaps due to the increased permeability of the vascular walls in a lung that has been compressed for a long time.

When the process is over, we close the little opening with a bit of sticking-plaster or with iodoform collodion. A regular surgical dressing is scarcely ever necessary.

If the exploratory puncture has shown a purulent effusion, we can first evacuate the pus by puncture if the vital indication exists; but a permanent cure from tapping is exceptional. The pus almost always reappears. Empyema is like an abscess, which cannot be cured until a permanent, free exit for the pus has been provided. We must, therefore, not only remove the pus, but institute drainage of the pleural cavity. The best method, and the one which is almost exclusively practiced in the surgical wards in Erlangen, is by incision of the pleural cavity, preceded by resection of a portion of a rib for the purpose of completely emptying out the pus, and obtaining a better final result. This comparatively simple method gives results so good and so entirely satisfactory that the employment of other methods is entirely unnecessary. We nevertheless concede that the so-called Bülau's siphon drainage gives very good results, especially in early and benign cases. It consists in the introduction of a rubber tube into the pus cavity through a large trocar cannula, and the provision then for continuous drainage through the establishment of siphonage. The method has the advantage of entirely avoiding a pneumothorax and of not requiring narcosis. In many cases, however, the procedure is found insufficient, so that thoracotomy must be performed later. The reader is referred to surgical text-books for the details of the above-mentioned methods, and of some others of a like nature, as well as their practical application.

In treating the chronic, contracted pleurisies with thickening, but without fluid effusion, methodical respiratory efforts, "lung gymnastics," are of use. Besides these, we should strengthen the general conditions as much as possible. We should advise the patient to breathe deeply, and prescribe cold sponging of the chest daily. Inspiration of compressed air by means of a pneumatic apparatus is often accompanied by good results. Well-to-do patients, who have had a severe pleurisy, should be sent to a suitable climatic health resort.

CHAPTER II

PERIPLEURITIS

Under the name of "peripleuritis" Wunderlich was the first to describe a rare form of disease, consisting of an inflammation of the connective tissue between the costal pleura and the ribs, and terminating in the formation of an abscess. Such cases have since been repeatedly observed, and all were characterized by the absence of any discoverable ætiological factors. There is neither a previous injury nor a primary disease of the ribs or the pleura. Nevertheless, the cause must be sought in an invasion of micrococci, which excite the suppuration. A knowledge of the particulars, however, can only be gained from future investigations. It is as yet undetermined whether peripleuritis can be regarded as an independent disease or not.

The disease occurs chiefly in men. It usually begins suddenly with a chill, and runs its course with quite a high fever. In pronounced cases the local symptoms have the greatest similarity to those of an empyema, but the greater protrusion of the chest wall is striking. The ribs are crowded apart by the abscess, and there is often spontaneous rupture externally, scarcely ever into the pleura. Percussion gives no symptoms of displacement of the neighboring organs, a distinguishing point from empyema. It is of diagnostic significance that we can often discover normal lung tissue containing air below the abscess. The mobility of the lower border of the lung is also usually retained, contrary to what is the case in empyema. Another important sign was first brought to notice by Bartels: the wall of the abscess relaxes on inspiration and becomes tense on expiration. We may also mention that acute nephritis has often been observed among the complications.

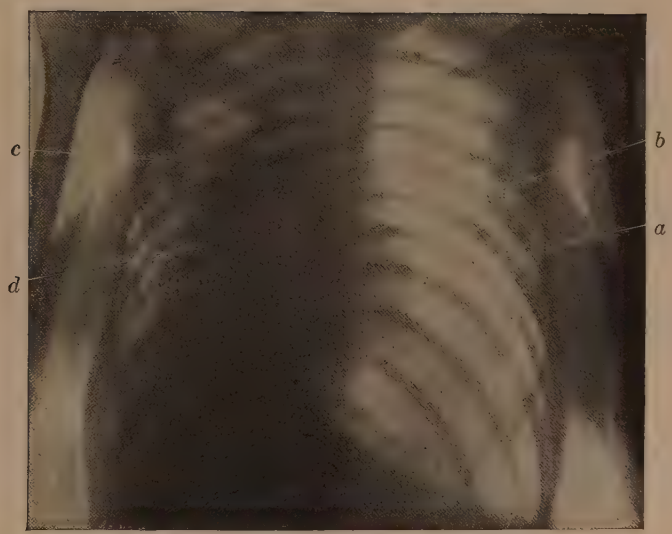
From these points we may be able to make the diagnosis during life, at least in many cases. The prognosis is quite unfavorable, but recovery does occur. The treatment can be only operative.

CHAPTER III

PNEUMOTHORAX

(*Pyopneumothorax. Hydropneumothorax.*)

Ætiology.—Pneumothorax—that is, a collection of air or gas in the pleural cavity—arises, in an overwhelming majority of cases, from the penetration of air into the pleural cavity through an opening in the pleura. The opening may be in the external chest wall from a penetrating wound of the chest or an empyema operation, or it may be in the pulmonary pleura. Pneumothorax is by far most frequently associated with phthisis, when a cavity lying beneath the pulmonary pleura perforates into the pleural cavity. This is more apt to happen in comparatively acute phthisis than in very chronic forms, because the extensive adhesions and contractions in the latter hinder



RADIOGRAM OF A RIGHT-SIDED PNEUMOTHORAX WITHOUT MUCH PLEURITIC EXUDATE. *a*, pneumothorax; *b*, compressed right lung; *c*, left lung infiltrated with tuberculosis; *d*, the heart crowded over to the left.

Note the lighter area corresponding to the large air space. In the left portion of this is the shadow of the lung, which is entirely compressed against the vertebral column. The heart and mediastinum are pushed over to the left side and the diaphragm strongly crowded downward. The left lung, particularly in its upper lobe, is studded with numerous tuberculous infiltrations.

its development. It usually appears in quite far-advanced cases, but it may sometimes arise with but slight changes in the lung.

Pulmonary gangrene or abscess, as well as phthisis, may cause pneumothorax by perforation into the pleural cavity. It may also arise from the rupture of an empyema into the lung. In some cases a perforation of the œsophagus or stomach into the pleura, as in gastric ulcer, has been observed, with the formation of pneumothorax.

The development of this condition from severe injuries, as from laceration of the previously healthy lung, without injury to the chest wall, is rare. Forced respiratory movements, associated with physical exertion, seem especially capable of exciting such a process. We have ourselves seen pneumothorax develop suddenly in a previously healthy woman while hanging out her washing, and another time in a young man during very labored rowing. Both cases recovered rapidly and completely.

All the last-named causes, however, are far less important than pulmonary tuberculosis. We should mention that in phthisis, too, there is sometimes a definite exciting cause—severe coughing, vomiting, or muscular exertion—which may favor the development of the pneumothorax.

Many authors maintain that, by decomposition of a putrid pleuritic effusion, gas may be produced, and thus we may have pneumothorax; but such an event is extremely rare, if it ever happens.

Pathological Anatomy.—On opening the pleural cavity a part of the air usually rushes out, sometimes with an audible noise. We then look into a large cavity filled with air, and find, in total pneumothorax, the lung completely retracted and lying compressed against the vertebral column. If, however, the air fills only a part of the pleural cavity, as a result of extensive adhesions of the pleuræ, we speak of a circumscribed or sacculated pneumothorax. The amount of air contained in the pleural cavity may reach 2,000 c.c. The pressure which it is under is almost always positive—on an average 5 or 10 c.c. of water.

In the cases of pneumothorax arising from perforation of the pulmonary pleura we can usually make out the point of perforation in the lungs. This is more frequently situated in the upper lobe than in the lower. Sometimes it is already grown over or is covered by a layer of fibrin, and can no longer be found. The opening is usually quite small, but it may reach the size of a ten-cent piece. Left-sided pneumothorax seems to be somewhat more frequent than right-sided.

The pleura itself is only rarely normal. Usually agents of inflammation have entered it with the air, and hence it is found in a state of inflammation. A part of the cavity is then filled with effusion. This is usually wholly purulent—pyopneumothorax—or seropurulent, but it may even be serous or serofibrinous—seropneumothorax, or hydropneumothorax.

The neighboring organs, especially the heart and liver, are found pushed out of their normal position, as in large pleuritic effusions.

Symptoms and Course.—The onset of pneumothorax (we speak in what follows especially of pneumothorax in connection with pulmonary tuberculosis) is quite often made known by a sudden pain, usually associated with an increase of the dyspnœa and of the general symptoms. There is sometimes collapse. The temperature sinks below normal, the pulse rises to 140 and

over. The patient looks pale and cyanotic. He usually sits upright or is in a half-sitting position in bed, either more on the affected side, in order to use the normal lung as much as possible for breathing, or more on the sound side on account of the tenderness. If the pneumothorax has come on as a result of the rupture of an empyema into the lungs, there is at the same time a very abundant expectoration of pus.

Although in many cases the symptoms mentioned lead to a suspicion of pneumothorax, yet a positive diagnosis can be made only after a physical examination.

Inspection gives a very marked distention of the affected side. The intercostal spaces are flattened, or even convex. In some cases, as we have ourselves noticed, there is a marked elastic "air-cushion feeling" on palpating the intercostal spaces. On respiration, the affected side is almost entirely motionless, while the excursions of the other side are the more marked. The displacement of the heart is often evident from the visible displacement of the apex beat.

Percussion gives over the pneumothorax a remarkably loud, full note, abnormally deep, but usually not tympanitic, on account of the tension of the walls. It is especially important to note that this resonance extends beyond the normal limits of the lung on the right down to the seventh or eighth rib, and on the left to the fifth or sixth rib, and sometimes even to the edge of the thorax. The lower limit of this abnormally loud percussion resonance shows no change of position with respiration.

The displacement of the neighboring organs can also be made out by percussion. With right-sided pneumothorax we find the lower border of the liver dullness abnormally low, and the left border of the cardiac dullness pushed over to the anterior axillary line. In left-sided pneumothorax the cardiac dullness is usually entirely absent from its normal place, and is found instead to the right of the sternum. The left lobe of the liver is pressed downward, and in the "semilunar space" we find, instead of the normal tympanitic resonance, the same deep, loud, and usually nontympanitic note as in the upper parts of the thorax.

Upon auscultation we are struck by the entire absence of respiratory murmur. This is in special contrast to the clear resonance on percussion. In other cases, however, we hear a number of metallic sounds, at least in many places and at many times, which are very characteristic of pneumothorax. First among these is amphoric, metallic respiration. This arises in open pneumothorax (*vide infra*) from the direct passage of the air in and out, but in all other cases it is the ordinary respiratory murmur, which has acquired a metallic timbre from resonance in the pneumothorax. In an analogous way arise the metallic-sounding râles ["metallic tinkling"], and the metallic resonance of the cough and voice. Heubner has devised a particularly beautiful and practically important method for demonstrating the metallic sound in pneumothorax. If we strike lightly on a pleximeter with a little rod, usually the handle of a percussion hammer, while we auscult near it—"rod percussion"—we very often hear quite a distinct high metallic sound.

The vocal fremitus over a pneumothorax is usually diminished, but it may be felt in spite of quite a large collection of air.

A number of special physical signs are found if a purulent or serous effu-

sion be added to the pneumothorax. In the first place, the resonance is thereby rendered dull, to a greater or less extent, in the lower parts of the chest. The boundaries of the fluid by percussion show a very evident change with the patient's change of position, because the fluid in pneumothorax can move easily in all directions. Inasmuch as the shape of the remaining air space must change correspondingly, there is not infrequently a change in the pitch of any metallic sounds which may be produced, whether by percussion or respiration, varying with the posture of the patient (Biermer's change of note). In many cases, if the ear is applied to the chest wall there is heard, on every motion of the fluid, produced, e. g., by gently shaking the patient, a metallic gurgling sound, so-called Hippocratic succussion.

Forms of Pneumothorax.—According to the condition of the perforation during life, we distinguish three kinds of pneumothorax (Weil). We speak of an "open pneumothorax," if the point of perforation remains open, so that the air on respiration constantly passes in and out of the pleural cavity. If the perforation is completely closed, we have a "closed pneumothorax." The third and most frequent form is the "valvular pneumothorax," in which air enters the pleural cavity at each inspiration, but on expiration there is a valvelike closure of the perforation, and thus the air cannot escape again; but as soon as the pressure in the pleural cavity increases so that no more air can enter it on inspiration, the valvular pneumothorax becomes closed. In open pneumothorax the pressure in the pleural cavity must be the same as the atmospheric pressure. A positive pressure in the pleural cavity can exist only in a closed or a valvular pneumothorax.

A clinical diagnosis of the form of pneumothorax is not always possible, and has usually no great practical importance. The very loud, metallic, amphoric respiratory murmur, which may be heard in open pneumothorax, must be mentioned, and Wintrich's change of pitch (see page 295) can sometimes be heard in this form. It is worthy of mention that symptoms of displacement of the neighboring organs must also arise in open pneumothorax. The predominant atmospheric pressure here is positive in contrast to the negative pressure in the other pleural cavity, and to the normal negative pressure which previously acted on the upper surface of the diaphragm. The compression of neighboring organs is, however, never as marked in open pneumothorax as in the closed variety. In the latter, the internal pressure becomes decidedly higher with the addition of a pleuritic effusion. For this reason, a marked protrusion of the affected side, and great displacement of the heart and liver, speak most strongly against an open pneumothorax. The percussion note is not rarely tympanitic in open pneumothorax because of the slighter tension of the chest wall; in closed pneumothorax, on the other hand, the percussion note is almost invariably nontympanitic. Some authors have tried to find a point of distinction for the different forms of pneumothorax in the composition of the gas in the pleural cavity, but the results of chemical analysis are still contradictory. According to Ewald, we find in open pneumothorax not over five per cent of carbonic acid and about twelve to eighteen per cent of oxygen; in closed pneumothorax, however, fifteen to twenty per cent of carbonic acid and ten per cent at most of oxygen. If in an open pyopneumothorax or hydropneumothorax the point of perforation lies below the level of the fluid, there sometimes arise on every inspiration metallic sounds, since

the bubbles of air drawn in rise and come up through the fluid—"the water-pipe sound," "metallic tinkling." A peculiar sipping and short snapping sound on inspiration, heard by us in a few instances, seemed to point directly to the existence of a valvular pneumothorax. In valvular pneumothorax, the intrathoracic pressure often attains the highest degree, and therefore results in very marked manifestations of displacement.

Course of the Disease.—In many cases the occurrence of pneumothorax causes such a high degree of respiratory disturbance that death ensues in a few hours or days. In other cases the patient improves, and may feel quite well for a long time in spite of the condition. We have ourselves frequently observed patients who passed the entire day out of bed with scarcely any inconvenience. I know of several cases in which the condition finally became completely stationary, and in which the patients went about with their pneumothorax for months or even years (!) without any symptoms. Usually, however, the disease which gives rise to the pneumothorax, most often pulmonary tuberculosis, leads in a shorter or longer time to death. Sometimes, again, the patient recovers. This almost always occurs in those rare cases above mentioned, when a healthy lung has been torn, but sometimes, also, in cases of pulmonary tuberculosis. The healing usually takes place in this way, that the air is first replaced by a fluid effusion, and then the latter is gradually absorbed, but the air may also be directly absorbed in whole or in part. It depends upon the origin of the lesion, then, and upon the intensity of the underlying disease, whether the recovery is permanent or not.

Diagnosis.—The diagnosis of pneumothorax is usually easy with careful examination. The most striking feature is usually the absence of the respiratory murmur, despite the tympanitic percussion note. When the attention is attracted by this sign, search is then to be made for the other sign. An X-ray examination may prove of great value in doubtful cases. It is sometimes very difficult to make a differential diagnosis between very large cavities and a saccular pneumothorax. We may mention as the chief points in distinction: a cavity is apt to be situated in the apex, pneumothorax in the lower part of the thorax; over a cavity the chest wall is often sunken in, over pneumothorax it is prominent; the vocal fremitus is usually marked over a cavity, weak over pneumothorax. Signs of the displacement of organs point to pneumothorax, as does distinct succussion, while a metallic respiratory sound and metallic resonance on rod percussion may also be heard over large cavities with smooth walls. Besides the above signs, we should also carefully regard the general course of the disease. On careful questioning we shall almost always find that the severe symptoms depending upon the development of a pneumothorax set in with more or less suddenness.

Treatment.—Whenever pneumothorax has developed we must seek to obtain perfect quiet for the patient as well as diminution of his sufferings, by means of morphin given subcutaneously or internally. The result of the administration of this drug, in a sufficient though careful dosage, is often very satisfactory. We must also stimulate cardiac activity, if it is impaired, by means of strophanthus, camphor, and the like. In cases presenting marked pressure symptoms and severe dyspnœa, we may attempt to remove some of the air in the pleural cavity by aspiration with a hollow needle, and, by this means, to relieve the dyspnœa. As a rule, however, we wait to see to what extent

the condition will improve spontaneously. If there is an abundant exudation it is always useful to puncture and remove the fluid, repeating the operation if necessary. If the exudation is purulent, the treatment is precisely the same as for empyema. In hopeless cases of far-advanced tuberculosis, we should limit our efforts to purely symptomatic treatment, or to the simple evacuation of the pus by puncture.

CHAPTER IV

HYDROTHORAX. HEMATOTHORAX

1. **Hydrothorax.**—We term the occurrence of a serous transudation into the pleural cavity, independent of an inflammation of the pleura, hydrothorax, or thoracic dropsy. The cause of hydrothorax is in rare cases a local hindrance to the outflow of venous blood or lymph from the thorax, as in compression of the veins or of the thoracic duct by tumors; but in the great majority of cases the hydrothorax is part of a general dropsy, occurring especially in cardiac or renal disease. Hydrothorax is often first developed after marked œdema of the subcutaneous cellular tissue and ascites, but it may sometimes be one of the first symptoms of dropsy, particularly in renal cases. It is usually bilateral, but it is often unilateral, or at least much greater on one side than on the other. The pleura itself is normal or else œdematous. We often find it traversed with a network of dilated lymphatics. The serous fluid in hydrothorax is distinguished from an inflammatory serous effusion (compare above with page 355) by the smaller amount of albumen in it, and a correspondingly low specific gravity (usually below 1.018), by the scanty number of cell elements (aside from the frequent admixture of exfoliated endothelial cells), and by the absence of or the slight tendency to spontaneous coagulation.

The clinical importance of hydrothorax lies in the hindrance to respiration which it causes. As a result of this the hydrothorax may be regarded in many cases, especially in renal disease, as the chief cause of death. Its demonstration comes from the physical examination, which, in general, gives dullness, diminished vocal resonance, and dislocation of neighboring organs, the same as in pleuritic effusion. We desire to emphasize also the bronchial respiration from compression in hydrothorax, which is often very loud, although at the same time usually high and sharp, and which may even give rise to a confusion with pneumonic infiltration in the lungs. This very loud respiratory murmur, contrasting with that of pleuritic effusion, is explained by the normal condition of the lungs and the absence of all adhesions. For the same reason, too, the change in the boundary of the dullness, as a result of the patient's change of position, is usually more marked in hydrothorax than in pleuritic effusion. In the case of a right-sided hydrothorax, the shifting of the fluid is generally most readily determined in front. In the sitting posture, there is, anteriorly, an area of dullness above the upper border of liver dullness, which disappears in the recumbent posture; but the change in the extent of percussion dullness, with change of position, can often be made out plainly enough in the back. We often hear a few fine crepitant râles over the hydro-

thorax, which arise in the retracted and partly atelectatic lung. The main factor, however, in distinguishing hydrothorax from a pleuritic exudation is, in every case, the consideration of the primary disease. It should also be noticed that hydrothorax is usually bilateral, while pleuritic exudation is usually unilateral. Yet, on the other hand, we occasionally find a much larger collection of fluid in one pleural cavity than in the other in hydrothorax.

Treatment is directed, first of all, to the primary disease. If we succeed in strengthening and regulating the action of the heart, or in restoring the secretion of urine, the hydrothorax often disappears with the other dropsical symptoms. If the dyspnœa caused by it reaches a dangerous degree, we often see great relief from aspirating the fluid. The nature of the underlying condition, of course, renders the benefit in many cases only transitory.

2. **Hematothorax.**—Effusions of blood into the pleural cavity (hematothorax) arise most frequently from traumatic lacerations of blood vessels, rarely from the bursting of an aneurism of the aorta into the pleural cavity, from erosion of an intercostal artery in caries of the ribs, from the rupture of a cavity into the pleura in phthisis, if it simultaneously opens a blood vessel, etc. In many such cases a typical exudative pleurisy follows the effusion of blood. The physical signs are the same as in other pleural effusions. Severe dyspnœa may demand the removal of the blood by puncture, or even by an incision.

CHAPTER V

NEW GROWTHS OF THE PLEURA

The majority of new growths occurring in the pleura are of a secondary nature. We sometimes find single metastatic nodules of cancer in the pleura after primary carcinoma of other organs, especially of the mammary gland and the lungs, but most carcinomata of the pleura arise from primary carcinomata of the lungs and are due to a direct invasion of the pleura by the new growth.

Of the primary new growths of the pleura, the rare round-celled sarcoma must be mentioned, and in addition, above all, there is the endothelial carcinoma, first described by E. Wagner. This develops, *de novo*, in a diffuse manner, from a proliferation of the endothelial cells of the lymphatics and the connective tissue. Metastases occur in the lungs, in the lymph-glands, in the liver, in the muscles, etc.

Single secondary nodules of cancer in the pleura cause no special clinical symptoms, but the cases of diffuse cancer of the pleura as a result of primary cancer of the lungs are important, inasmuch as the symptoms of disease of the pleura often quite predominate over the pulmonary disease. The dullness is intense, the respiratory murmur and the vocal fremitus diminished. In one such case we saw a proliferation of the cancer upon the ribs in front so that there was externally a very marked circumscribed swelling. The character of the sputum is the only thing that can give us definite information as to

the origin of the new growth in the lungs (see the chapter on cancer of the lungs).

Primary endothelial carcinoma of the pleura runs a course similar to chronic pleurisy. As we sometimes find a coexisting fluid effusion in the pleural cavity, displacement of the neighboring organs may occur. The affection goes on for a long time without fever, or with slight and irregular elevations of temperature. There are some forms of malignant, rapidly growing tumors that lead to death in a few months. Most cancers of the pleura are associated with severe pain.

The diagnosis of new growths in the pleura can usually be made, if at all, only in the more advanced stages of the disease. At first almost all the cases are regarded as simple or tuberculous chronic pleurisy. There are, however, certain striking features of pleural new growths, viz., the unusual extent of the percussion dullness (much more marked anteriorly than posteriorly, for example), the absence of the general earmarks of tuberculosis, the appearance of slight œdema of the chest wall, of one arm, and the like. The examination of the fluid obtained by an exploratory puncture furnishes important evidence. It is usually hemorrhagic. The red blood cells occasionally show degenerative changes, giving the effusion a brown color. The microscopical examination reveals a striking number of endothelial cells that show variations in size and shape. They have often undergone fatty degeneration, so that the occurrence of numerous cells containing fat granules or even of free fat globules, should always lead one to think of a new growth. In rare cases more coherent fragments of tumor tissue may be microscopically demonstrated in the aspirated fluid.

The prognosis is absolutely unfavorable, and the treatment purely symptomatic. At most, we may try X-ray treatment, or the subcutaneous injection of atoxyl or cacodyl.

CHAPTER VI

MEDIASTINAL TUMORS

In the anterior mediastinum, in quite rare cases, extensive new growths occur, which are of importance on account of their severe clinical symptoms. The point of origin for the tumor is either the mediastinal lymph-glands, or the connective tissue, or sometimes the remains of the thymus gland. In their anatomical character the tumors are almost always sarcomata, usually lymphosarcoma, rarely alveolar sarcoma. They usually occur in youth or middle age, and are somewhat more frequent in men than in women. The special ætiological factors are unknown. In some cases an injury is stated to be the cause of their development. Sometimes the mediastinal tumors are only a part of a general pseudo-leukæmia or a lymphatic leukæmia.

The first clinical symptoms are usually of a very indefinite nature. The patient complains of general languor, headache, pain in the chest, and slight difficulty in breathing, and only gradually do severe subjective and objective symptoms develop in the chest.

The symptoms are in part due directly to the tumor, but in larger part they are symptoms of compression from the gradually increasing pressure of the tumor on a number of neighboring organs.

The pain in the chest, which is located chiefly in the sternal region, and is associated with a marked feeling of oppression, may be very severe. It sometimes shoots into the lateral portions of the chest and into the arms, showing pressure on the brachial plexus.

The dyspnœa may finally increase to an extreme degree. A patient with lympho-sarcoma under our observation could, in the last days of her life, breathe only while standing. The dyspnœa is due to a compression of the heart and lungs, and sometimes to actual stenosis of the trachea or a primary bronchus. Paralysis of the dilators of the glottis may also occur from a pressure paralysis of the recurrent nerves. Paralysis of one vocal cord has been repeatedly observed. In the case mentioned above a marked goiter developed, as a result of vascular stasis, which further increased the dyspnœa by pressure on the trachea. A hydrothorax from local venous stasis may also aid in increasing the dyspnœa.

Pressure on the œsophagus, and disturbances of deglutition due to it, are rare. Pressure on the vagus nerve and the sympathetic sometimes causes anomalies in the rate of the pulse—either marked acceleration or slowing of the pulse. If the sympathetic is involved there is inequality of the pupils. In some cases, by pressing on the tumor, an artificial dilatation of the pupil can be excited at will. By pressure on the vessels, especially on the superior vena cava, the subclavian vein, etc., œdema and cyanosis may arise in the corresponding parts of the body.

Objective examination of the chest gives a marked diffuse prominence of the sternal region in a part of the advanced cases; in other cases this swelling is absent. The discovery of an abnormal dullness in the anterior part of the chest is of diagnostic importance; this usually joins the cardiac dullness on the left, and on the right it extends a varying distance beyond the right border of the sternum. The heart is often pushed somewhat to the left. We heard over the pulmonary artery in our case a marked systolic murmur, caused by compression of the vessel. A dissimilarity of the pulse on the two sides is not infrequent.

The diagnosis of a mediastinal tumor is usually possible in cases with well-marked symptoms, but in other cases it is difficult and uncertain. In many cases X-ray examination gives decisive results. The differentiation between mediastinal tumors and aneurisms of the aorta (*q. v.*) sometimes presents great difficulty. Tumors may also be confounded with abscesses in the anterior mediastinum.

The prognosis is in all cases absolutely unfavorable. The disease terminates fatally, sometimes after a duration of six months or a year.

The treatment can be merely symptomatic. Internally we may try iodid of potassium or arsenic. In addition to these, X-ray treatment is to be principally considered (compare with the chapter on Leukæmia and Pseudo-leukæmia). In the last stages of the disease we must try to alleviate the patient's great distress by narcotics.

CHAPTER VII

ACTINOMYCOSIS OF THE THORACIC CAVITY

Bollinger and others have described a peculiar tumor affecting the jaw-bones of cattle, and occasioned by the presence of a special form of fungus, known as the actinomyces or ray fungus. More recently a class of diseases has been studied in human beings, occasioned by the same fungus (Ponfick, Israel, and others). These diseases may, as in cattle, affect the jaws, the floor of the mouth, and the neck; but in these cases they are mainly of surgical interest. The actinomycotic diseases of the internal organs, however, possess a great clinical importance; and, inasmuch as the lungs and pleura are the most frequently affected organs, it will be well to present briefly here the most important facts which have as yet been learned with regard to actinomycosis.

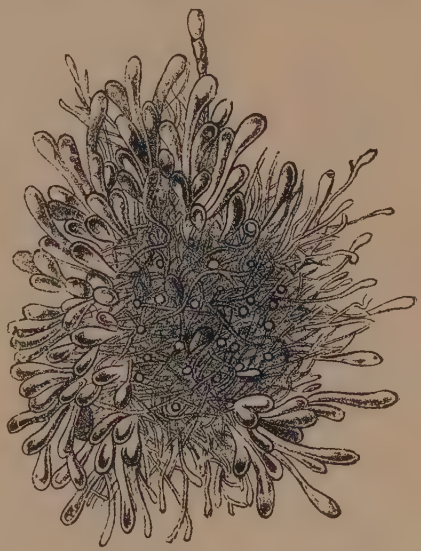


Fig. 59.—Masses of actinomyces. (From Johne.)

The botanical position of the actinomyces is not yet definitely settled. According to recent investigations, there is quite a close relationship between actinomyces and the tubercle bacillus. In its growth the fungus forms small or moderate-sized gray or sulphur-yellow nodules which may be distinguished with the naked eye in the pus of the diseased tissue (see below), and which upon microscopic examination resolve themselves into a tangle of mycelium. It is an especial characteristic that many of these mycelia bear on their ends a club-shaped swelling. These are placed for the most part like radii on the periphery of a nodule, and so surround the entire mass like a circlet of rays (see Fig. 59).

In nature actinomyces seems to appear especially upon plants, for example upon the beard of spikes of wheat. Thus is explained the frequency of infection in the plant-eating cattle, and a similar direct infection seems occasionally to be possible in man. It is worthy of note that the fungus seems to locate itself often in carious teeth. Thus apparently arises the above-mentioned disease in the buccal cavity; while, on the other hand, the fungus may be carried from its nidus in the mouth by inspiration into the respiratory tract, or by swallowing into the *primæ viæ*. Of course it may also be directly swallowed or inhaled. Primary intestinal actinomycosis generally begins in the cæcum or in the appendix.

Wherever the fungus fastens itself in the body it occasions first a new growth of granulation tissue, which has a tendency to break down into a whitish or brownish pasty mass. The brown color is occasioned by the hemor-

rhage which frequently occurs. Very often actinomycosis goes on to suppuration; in this, of course, secondary infection by pus germs (pus cocci) may also play a rôle. Of special importance is the tendency of the disease to extend from the lungs to the pleura and from the pleura to the peripleuritic connective tissue, and still further to the wall of the thorax. Thus arise not only extensive abscesses and wide-branching fistulous tracts, but also a very characteristic, extremely tough cicatricial infiltration of the affected part. Not infrequently there is at last a perforation reaching the outer surface of the body.

The entire process, as a rule, is slow and insidious, but constantly progressive. The symptoms consist at first in slight thoracic discomfort, pain, cough, and expectoration. Physical examination will often detect changes in the lungs, but the correct interpretation of the signs found is, of course, at first difficult, if not impossible. Some cases are at first regarded as pulmonary tuberculosis or tuberculous pleurisy, until the appearance of the peculiar infiltration of the skin over the area of dullness awakens a suspicion of actinomycosis. The diagnosis may be made certain by exploratory puncture. Actinomycosis granules have also been frequently demonstrated in the sputum. The more the disease spreads the greater is the distress. Usually there is hectic fever, which may assume a pyæmic character if there is extensive suppuration. The patient gradually loses flesh, and in repeated instances amyloid degeneration of the liver, spleen, and kidneys has been observed. If a focus breaks into a pulmonary vein, the disease may be developed by metastasis in other internal viscera. Moreover, there may be a direct extension of the disease to the pericardium, or through the diaphragm into the peritoneal cavity.

The treatment can be only symptomatic, unless the diseased spot can be reached by operation, and then the treatment becomes surgical. Permanent cure has so far been attained in but rare instances. The continued use of iodid of potassium or of sodium has occasionally been of decided benefit.

III. DISEASES OF THE CIRCULATORY ORGANS

SECTION I

Diseases of the Heart

CHAPTER I

ACUTE ENDOCARDITIS

(Endocarditis verrucosa. Endocarditis ulcerosa)

Ætiology.—Organized excitants of inflammation of different sorts, which circulate in the blood, may settle on the endocardium, especially on the valves of the heart, and there give rise to an acute endocarditis. Endocarditis, therefore, in its ætiological relations, is not to be regarded as a single disease. Pathogenic microörganisms have been injected into the blood (the *Streptococcus pyogenes, Staphylococcus aureus,* and others), and in this way an artificial endocarditis has been set up in animals. The experiments are more apt to succeed if the valves or the inner coat of the vessels have been subjected to some slight injury before the injection, thus promoting the settling of the germs upon them (Orth and Wyssokowitsch, Ribbert). Most cases of endocarditis in man, both the vegetative and ulcerative forms, appear to be caused by the *Staphylococcus pyogenes aureus.* Streptococci are much less frequent than staphylococci, and it is only in rare cases that endocarditis is occasioned by the pneumococcus, gonococcus, and perhaps the diphtheria bacillus.

In accord with these statements is the fact that in human beings acute articular rheumatism, which is probably to be regarded as a staphylococcus infection, is that one of the infectious diseases which is especially apt to have acute endocarditis as a frequent and important complication. The staphylococcus infection need not appear, however, in the first place, as a typical acute polyarthritis. An acute endocarditis may appear as a sequel of many cases of apparently primary pleurisy, tonsillitis, or other disease. Indeed, it is sometimes impossible to determine the gate of entry of the infection, and the whole disease takes the shape of an apparently primary endocarditis. In such cases the nature of the disease is made evident by the later appearance of multiple articular swellings. We also regard as allied to polyarthritis certain forms of infection called the "acute hemorrhagic diseases" (e. g., purpura rheumatica), and also chorea (which see). It is therefore not surprising that acute endocarditis is not infrequently observed in association with these affections.

There are other acute infectious diseases with which acute endocarditis may be associated. Probably in these it is usually the result of a secondary mixed

infection, generally with the staphylococcus. This explains the appearance of acute endocarditis in scarlet fever, smallpox, diphtheria, measles, and typhoid. Not infrequently mild cases of acute endocarditis, without clinical significance, are found in association with primary tuberculosis and ulcerating carcinoma. Again, acute and chronic nephritis may sometimes occasion a development of endocarditis. An especially important rôle in producing endocarditis is displayed by grave septic and pyæmic conditions (*vide supra*). In these cases the acute endocarditis appears as one symptom, but it is often so prominent that the whole attack is called endocarditis (*vide infra*). In such severe diseases we have often to do with a streptococcus disease, although it may be a staphylococcus infection of especial virulence. Finally, gonorrheal endocarditis must be mentioned. Endocarditis as a sequel of gonorrhea is fortunately very rare. It is often an actual gonococcus infection, but in other instances due to a mixed infection.

Finally, we have still to mention the important fact that acute endocarditis quite frequently develops on the soil of an already existing chronic endocarditis —the so-called acute recurrent endocarditis. In women, pregnancy and the puerperal state sometimes seem to give the occasion for a recrudescence of the endocarditis.

Pathological Anatomy.—We usually distinguish an *endocarditis verrucosa*, with the formation of large or small papillary nodules on the endocardium, and an *endocarditis ulcerosa* (*endocarditis diphtheritica*), with ulcerations as a result of the destruction and wasting away of the superficially necrosed tissue. The malignant, invariably fatal form of severe septic endocarditis is chiefly ulcerative endocarditis. Endocarditis verrucosa is the milder form, which is seen especially in acute polyarthritis and allied diseases, but we cannot draw either a sharp anatomical or a sharp clinical distinction between the two, since malignant cases of endocarditis verrucosa are also observed.

The endocardial growths are usually situated on the valves, especially on their edges of closure. More rarely we find them on the chordæ tendinæ and on the endocardium of the ventricle and auricle. In the mildest cases they are scarcely as large as the head of a pin, but in severe cases they may increase to quite large warty and glandular masses. Microscopically, the base of the nodule consists of a newly formed vascular tissue, infiltrated with small cells, which on its surface changes to a granular, coagulated mass. This last is formed partly of coagulated albumen, dead cells, and fibrin deposited from the blood, and partly of micrococci (*vide supra*). The micrococci are found without exception in all cases of ulcerative endocarditis—having been first discovered by Eberth. The endocardial ulcers arise from the destruction of the superficially necrosed nodules. If the thin valve in any place yields to the blood pressure, we have the so-called acute valvular aneurism. Complete perforation of a valve, and tearing off of fragments of a valve and of the chordæ tendinæ, are also seen.

The great majority of cases of acute endocarditis are confined to the valves of the left side of the heart—the mitral and aortic valves. Endocarditis on the tricuspid valve is seldom seen except as a secondary affection in old cases of heart disease. In a case of acute ulcerative endocarditis in a grown man seen by us, the process was confined exclusively to the tricuspid valve, and there were very many embolic abscesses in the lungs. This may be considered

a great rarity. In contrast to the ordinary localization of endocarditis we find fetal endocarditis most frequently in the right side of the heart.

Many other organs may be affected by the endocarditis, through embolism. In the benign endocarditis verrucosa, the masses of fibrin deposited on the irregularities of the valve furnish the embolic material. They cause large or small infarctions in the kidneys and spleen, embolic softening of the brain, etc. In the malignant, ulcerative forms, however, large numbers of bacteria get into the circulation at the same time with the necrotic masses of tissue which have been torn off. Here, then, we have to do not merely with simple mechanical obstruction but with infectious emboli. The emboli in ulcerative endocarditis, therefore, either give rise to embolic abscesses in the cardiac muscles, the kidneys, the spleen, the lungs, the retina, etc., or they result in hemorrhages, especially into the skin, but also into the kidneys, the brain, the retina, and the serous membranes. It is not yet known why in some cases abscesses are more frequent and in others hemorrhages. The two, however, may be combined. In general, we may suppose that the development of abscesses is everywhere connected with the presence of bacteria, mostly staphylococci, while hemorrhages may also arise from toxic influences; but again changes in the vascular walls caused by bacteria might give rise to hemorrhages. Embolic abscesses belong almost exclusively to the severe form of septic endocarditis. Hemorrhages are seen in this form, and also—without coexisting abscesses—in certain severe forms of endocarditis occurring in the course of acute rheumatism and allied diseases.

Clinical History.—Since acute endocarditis is not ætiologically a distinct disease, and since its clinical course is very different in different cases, it seems advisable to us to describe, in what follows, the most important varieties separately; but it must be expressly noted that the separate classes can by no means be sharply defined, and that there are many intermediate forms.

1. *Slight endocarditis verrucosa* is quite frequently found in the cadaver, without the slightest signs of any affection of the heart during life. The little papillary excrescences on the valves of the heart in phthisis, and carcinoma, whose ætiology has been explained above, are to be classed under this head.

2. The typical form of *benign acute endocarditis* is most frequent, clinically, in the course of acute articular rheumatism. It is much rarer in other infectious diseases (*vide supra*). In a few cases it is also seen as an apparently primary disease. Here there is a constitutional rheumatic infection which has taken place in some way, and which settles directly on the valves of the heart instead of in the joints (so-called primary rheumatic endocarditis). Careful questioning will sometimes reveal, or at least render probable, the place of infection (a mild case of tonsillitis or some slight external injury). Very often there will be later the symptoms of articular rheumatism. It is seldom that endocarditis as such is associated with special local disturbances, such as pain in the region of the heart, palpitation, and dyspnœa. Ordinarily the heart disease is discovered only by physical examination. The impulse of the heart in many cases is abnormally strong and diffuse; the pulse is accelerated, but strong, often somewhat jerky (*pulsus celer*), and usually regular, but sometimes a little irregular. Percussion at first shows no deviation from the limits of normal dullness. On auscultation, we hear at the apex, more rarely at the base, a loud blowing, systolic souffle. Diastolic murmurs are rare in

acute endocarditis. The pulmonic second sound is often accentuated. The physical signs referable to the heart are only slightly marked in many cases of acute endocarditis. This is understood if we remember that the occurrence of a heart murmur depends wholly on the localization of the endocarditis, on the development of some valvular insufficiency, etc.

Besides the direct symptoms pointing to the cardiac affection, the onset of an acute endocarditis is often, but not always, associated with fever, or, if fever be already present, with an increase of it, and with a slight aggravation of the general disturbance. Embolic processes may occur in the brain, the spleen, the kidneys, and the extremities, but they are comparatively rare. Sometimes a pericarditis develops as a result of the endocarditis (*vide infra*).

It is hard to make any accurate statements as to the duration of this form of endocarditis. The physical signs may last for days, or for several weeks. Complete recovery is possible, but in the majority of cases this variety passes into chronic valvular disease of the heart.

3. *Malignant Form of Acute Endocarditis—Septic Endocarditis.*—In these cases the endocarditis is only one aspect of a general septic infection (*vide* preceding chapter on septic affections). The severe general infection is therefore usually quite prominent. The objective signs in the heart are the same as in the preceding form, but more intense and extensive. The subjective cardiac symptoms, such as palpitation and distress, may be quite pronounced, but they may also be almost wholly absent in this form. The general condition, however, is usually bad. There is sometimes high fever with an irregular or intermitting course, but in many cases the fever is remarkably low in spite of quite severe constitutional symptoms.

The constitutional infection is very often manifested by the appearance of septic exanthemata or hemorrhages in the skin, sometimes in the mucous membranes (the conjunctiva and the soft palate) and in the retina. Secondary articular swellings often develop; they are of a serious character in the milder cases and purulent in the graver forms. Renal hemorrhages and acute hemorrhagic nephritis are quite frequent. Large emboli may also occur in the different organs in this as in every other form of endocarditis. Whether simple infarctions or metastatic abscesses form, depends on the kind of emboli.

The course and termination of the disease depend, above all, upon the virulence of the bacteria. There are comparatively mild cases of septic endocarditis which end after several weeks in a complete, or more often a relative, cure (with subsequent cardiac lesion), as well as malignant forms (so-called malignant ulcerative endocarditis), with a rapidly fatal course, or with a more protracted one that finally also ends unfavorably.

4. *The recurrent form of acute endocarditis* consists of an acute increase of the endocardial process, brought on by some exciting cause, in a patient already suffering from chronic endocarditis. The acute disease may show all the gradations from the mildest to the severest. The mild cases often run their course without any special symptoms. To this form we may frequently refer the more or less temporary elevations of temperature which we often see in patients with chronic valvular disease of the heart. In rarer cases the recurrent endocarditis comes on quite suddenly in the form of a severe acute attack. This sometimes seems to be clinically a primary, independent disease, especially if the previous chronic heart disease has up to that time caused no special symp-

toms. The patient has general malaise, headache, chills, and fever. The last may be quite high—104° F. (40° C.) and over—or moderate, varying between 100° and 102° F. (38° to 39° C.), or it may be entirely absent. In many cases it is intermittent, when the return of fever is often associated with a chill. The cardiac symptoms may be quite pronounced, but in this form, too, they may be obscure and indefinite. In the further course of the disease we meet with cutaneous hemorrhages, retinal hemorrhages, articular swellings, large renal hemorrhages, or typical hemorrhagic nephritis—in short, just the same general type of disease as in the other malignant forms of acute endocarditis. The course is rarely rapid, and often lasts for weeks. Severe cases almost always end fatally.

Diagnosis.—The diagnosis of an endocarditis, coming on secondarily in the course of articular rheumatism and other diseases, can be made only by a physical examination of the heart. We must therefore give constant attention to the condition of the heart in diseases which we know may give rise to endocarditis.

The diagnosis of the malignant form of endocarditis often causes great difficulty, especially if the patient is not seen until the later stages. It is confused with typhoid, meningitis, or acute miliary tuberculosis. Examination of the heart may furnish positive evidence, but sometimes, as we have said, there are no physical signs of cardiac disturbance, or the signs present are indefinite. Many a case of long-continued intermittent fever without demonstrable anatomical cause is finally discovered to be an acute endocarditis. A special diagnostic importance attaches to the secondary swelling of the joints, and also to the hemorrhages into the skin and retina, for these are much more infrequent in the diseases with which this form of endocarditis may be confounded. The acute hemorrhagic nephritis, too, in connection with the other symptoms, is, at least to a certain degree, characteristic of malignant endocarditis. A careful search for some ætiological factor is very important for diagnosis in all cases. For other points the reader is referred to the consideration of septic diseases (see page 133), where the importance of a bacteriological blood examination is also emphasized.

Prognosis.—In the description of the course of the disease we have already mentioned the prognosis of the different forms. The severe cases of acute endocarditis usually, and the cases of severe septic endocarditis always, end fatally. Here, however, the cause of death is to be sought rather in the accompanying systemic infection than in the endocarditis itself. In mild cases recovery is possible, but the process of repair is often so incomplete that chronic valvular disease of the heart develops from the acute endocarditis.

Treatment.—The chief requisite in the treatment of every endocarditis is as complete rest as possible for the patient. If ice is well borne, the continuous application of an ice bag to the cardiac region is of service. If there are signs of cardiac weakness, such as a small, rapid, and irregular pulse, we must employ cardiac stimulants, strophanthus, camphor, and, above all, digitalis. It must be confessed that the effect of these is not very great. If there are marked subjective symptoms (such as dyspnœa), narcotics, particularly morphin, are indispensable. Effort is made to combat the systemic infection by salicylate of sodium and similar remedies, including aspirin, phenacetin, and salipyrin. Quinin is usually entirely without effect, even when the fever is of an inter-

mittent character. Arsenic, however (by mouth or subcutaneously in the form of atoxyl or the cacodylate), seems to be useful, particularly in the more protracted cases.

[For remarks upon the alkaline treatment of rheumatism, see Vol. II. Attempts to cure septic endocarditis by specific sera or by injecting sterilized bacteria (preferably autogenous) have met with but limited success.]

CHAPTER II

VALVULAR DISEASE OF THE HEART

(Chronic Endocarditis)

ÆTIOLOGY

A LARGE number of cases of chronic valvular disease of the heart proceed from acute endocarditis. Hence the frequent statement in the history of chronic valvular disease that the patient has had articular rheumatism, once or many times. In a collection of 163 cases of undoubted valvular heart disease, we were able to ascribe 86 to a previous attack of acute articular rheumatism. Following the acute valvular endocarditis, which is often associated with this disease, marked thickening of the valves occurs, due to the growth of connective tissue. There are also changes in the way of contraction, adhesion, and finally often of considerable calcification. The unavoidable result of all these processes is that the altered valves are rendered incapable of fulfilling their physiological function of regulating the circulation. Inasmuch as the mitral valve is most often attacked by endocarditis when associated with acute articular rheumatism, we find mitral disease predominating among rheumatic valvular troubles; but lesions of the aortic valves, rheumatic in their origin, are by no means rare.

If we find a valvular lesion in a patient who has never suffered from articular rheumatism or chorea, we may, in some cases, be able to refer the origin of the valvular trouble to a previous attack of acute endocarditis, excited by one of the other causes above mentioned. Thus it is well known that acute endocarditis may occur in the course of scarlet fever, diphtheria, and typhoid fever, and eventuate in chronic disease, but this is, according to our experience, very rare.

In quite a large number of cases of heart disease, however, we cannot obtain a history of acute endocarditis. We have to do here with an endocarditis which is chronic from the start, which also leads gradually to thickening, contraction, adhesion, and calcification of the valves.

The causes of this chronic fibrous endocarditis are probably the same as of acute articular rheumatism, but in such cases they act from the start in a chronic manner. Perhaps often there may have been an incipient acute endocarditis, whose course was unperceived. We not infrequently learn from patients with chronic heart disease, who have never had an attack of acute articular rheumatism, that in former years they did suffer repeatedly from mild rheumatic pains, to which they paid little attention. Furthermore, it is

by no means exceptional to observe that such patients with a well-developed cardiac valvular lesion later on undergo one or more attacks of acute polyarthritis. Heart disease is also rarely associated with genuine chronic arthritis deformans. In other cases of valvular disease we must consider the possibility of other lesions, some infectious, some perhaps of a chemical and mechanical nature. Here belong, in the first place, those cases of cardiac disease which are associated with general arteriosclerosis (atheroma of the blood vessels). Indeed, atheromatous degeneration of the aorta seems sometimes to extend directly to the aortic valves, and thus cause a valvular lesion. Every ætiological factor favorable to general arteriosclerosis is, therefore, important in the ætiology of valvular disease. This includes advanced life, excessive physical labor, alcoholism, and genuine gout. Another important cause is syphilis. Of late years, since we have paid more attention to this last factor than formerly, we have observed many cases of valvular disease which were almost certainly of syphilitic origin. In particular, lesions of the aortic valves, when there is no evidence of other causation, must awaken our suspicion of syphilis. There remains to be mentioned the influence of chronic nephritis upon the development of cardiac valvular disease, although in the not infrequent cases in which chronic nephritis, particularly of the interstitial variety, is associated with chronic endocarditis, it is not always easy to determine whether both conditions are related in the way of cause and effect, or whether they are both secondary, and both alike the result of some third unfavorable influence. A hereditary predisposition to heart disease is not very frequent, but yet it can be made out with certainty in many cases. We have ourselves seen five members of the same family who have suffered from chronic heart disease, some from pure valvular disease and some from severe so-called idiopathic hypertrophy. Perhaps the very frequent occurrence of heart disease in many families is also connected with a special family predisposition to rheumatic affections, the occurrence of which predisposition cannot, in our opinion, be denied. Finally, a small number of cases of heart disease, especially in the right side of the heart, depend upon anomalies of development of the heart—congenital heart disease.

Valvular disease of the heart occurs at every age of life. The time of origin of most cases, corresponding in part to the occurrence of acute articular rheumatism, falls in youth and middle age, somewhere between eighteen and forty; but severe valvular disease is not infrequent even in children; while late in life the clinical picture of valvular disease is often confused by the simultaneous presence of general arteriosclerosis, pulmonary emphysema, or kidney trouble. Heart disease is said to be rather more frequent in the female sex than in the male. Women with heart disease not infrequently date their symptoms from pregnancy and the puerperium.

GENERAL PATHOLOGY OF VALVULAR DISEASE OF THE HEART

Every valve of the heart, in order to fulfill its physiological task, must, on the one hand, open perfectly at the right time in order to furnish a free passage to the blood current through the appropriate orifice, and must, on the other hand, close firmly and perfectly at the right time in order to make any

abnormal backward flow of blood impossible. In both relations the function of the valves may be disturbed by chronic endocarditis, the disturbance being the result of their anatomical changes. If the tips of the valves are shortened on their free edges by contraction, or if the complete unfolding of the auriculo-ventricular valves is hindered by a shortening of their chordæ tendinæ, the closure of the valve cannot be complete. At the moment when the closure of the valve is necessary a fissure remains open between its apices. We call this condition an insufficiency of the valve. On the other hand, the valves may lose their capability of free and sufficient separation from one another, as a result of thickening and calcification of the connective tissue, and also as a result of adhesions of the points of the valves with one another. At the moment when the blood current should pass freely through the open orifice, the valve remains a stiff, narrow ring, through which the blood must force its way—stenosis of the orifice. The changes in the valves are often of such a sort that they cause at the same time both an insufficiency of the valve and a stenosis of the orifice. The thickening and calcification of the valves in stenosis cause, as a rule, a valvular insufficiency at the same time; but an insufficiency, set up by a contraction of the edges of the valves, may occur without a coincident stenosis of the orifice.

The abnormal and injurious effect of a valvular lesion upon the circulation in the heart is felt in two directions. Either the obstacles to the circulation are increased in certain places, or there is a greater diastolic distention of certain portions of the heart. Both circumstances of course demand greater cardiac effort. If the heart were to labor with only the same degree of energy as under normal conditions, in spite of the increased resistance, or in spite of the greater distention, there would speedily be an impairment of circulation, incompatible with the continuance of life; for, if the increased resistance were not overcome, or if the abnormally distended cavity were not properly emptied, there would at once be a rapidly increasing congestion behind the diseased valve, and beyond it a constantly diminishing pressure. We can speak of a circulation of the blood only when, in a given interval of time, exactly as much blood is driven out of the heart as flows into it. The slightest difference in this regard would, in a very brief period, produce such a congestion of blood in the veins and such a diminution of blood in the arteries that the demand of the tissues for oxygen could no longer be satisfied, and death would be the necessary result. A normal circulation is maintained so long as the rapidity (the momentum) of the blood stream, as well as the amount of arterial blood, which flows into the organs in a given interval of time, is sufficiently great. Withal, the circulation must at any time be capable of immediate adaptation to any temporary increase in the demands of the organs, as, for example, when there is bodily exertion. The amount of blood flowing through the organs in a given time depends upon the degree of distention, the frequency of contraction, and the complete emptying of the left side of the heart. This amount ("the size of the circulation") may be diminished, and yet the circulation continue as such; but that the circulation can, in spite of the disturbance occasioned by a valvular lesion, still be maintained in a satisfactory manner is due to the capability of the heart of overcoming the obstacles to the circulation by means of increased work. It is one of the wisest contrivances in our organism, that the heart has control of a reserve fund of strength, which comes into action, if

need be, in a way to compensate as far as possible for any disturbance of the circulation. This explains why a man with valvular disease of the heart may be almost perfectly well for a long time, while the increased work of certain portions of his heart suffices to keep up an approximately normal circulation in spite of the existing valvular disease. We call a heart disease, in which there is at least no marked disturbance of circulation, a compensated heart disease.

The abnormal increase in functional activity of certain portions of the heart, associated with every cardiac lesion, and referable in every case, as we have said, either to increased resistance or to increased distention, results in a hypertrophy of those particular portions of the heart, just as in the case of any other muscle. The development of this hypertrophy may be explained by the fact that the increased labor involves a greater catabolism in the cardiac muscle, and this in turn is followed by an increased tendency to assimilation and growth of the muscle. The hypertrophy does not consist of an increase in thickness of the individual muscular fibers, but chiefly of an increase in number. The total bulk of the cardiac muscle increases, and thus its capacity for work naturally becomes greater. It goes without saying that increased nutritive processes and a large supply of nourishment for the heart are necessary to bring about such a hypertrophy, by which alone a compensation of the heart disease is possible for any length of time. Hence we find the secondary hypertrophy of the heart absent, or at least only imperfectly developed, in feeble individuals, especially in those who have suffered from some other chronic wasting disease besides the heart disease, such as phthisis or carcinoma. With this hypertrophy of the cardiac muscle, however, there is also associated a permanent dilatation of those heart cavities which have to take up increased amounts of blood during their diastole. To the extent to which this dilatation aids the circulation, it may be termed compensatory dilatation.

Although the compensatory processes in the heart may prevent for a long time any marked disturbance of the circulation, the already overburdened heart can no longer completely satisfy any excessive demands upon it, even in a compensated heart disease. Hence patients with a compensated heart disease are free from subjective disturbance only when they remain at rest, while the signs of a disturbed circulation usually become quite apparent on slight physical exertion.

The hypertrophied cardiac muscle can seldom supply permanently the abnormally great drafts made upon it. There finally comes a condition of "fatigue," of "cardiac insufficiency." The cause lies either in the increase of the valvular disease, so that the hindrance to the blood current caused by it can no longer be completely overcome, or in the fact that the nervous and muscular elements in the heart have their powers gradually impaired by a disturbance of circulation in the heart itself.

Very often a chronic myocarditis becomes associated with the valvular lesion (chronic endocarditis), and, naturally, further diminishes the working capacity of the heart. In consequence, the cardiac muscle loses its tone, the heart chambers can no longer completely empty themselves, and as a result of the stasis there is a gradually increasing filling up, until a condition of overdistention or of so-called congestive dilatation supervenes. In short, in every heart disease the moment may finally come when the capacity of the heart

has reached its limit, and hence the compensation of the heart disease ceases. The results of stasis now appear with increasing severity in the different organs, as we shall learn to recognize later on, and the patient finally succumbs to them, unless some intercurrent event puts an end to life.

After these general remarks, which will be understood better on reading what follows, we will pass on to the special description of the different forms of heart disease and their physical signs.

VARIOUS FORMS OF HEART DISEASE AND THEIR PHYSICAL SIGNS

1. **Insufficiency of the Mitral Valve.**—Mitral insufficiency is one of the most frequent forms of heart disease. It develops in acute or chronic endocarditis of the mitral valve, from contraction of the free edges of the valve or from shortening of the chordæ tendinæ. In rare cases it comes on from partial adhesion of the valves to the walls of the ventricle.

The closure of the mitral valve occurs normally at each systole of the left ventricle. It prevents the return of blood from the left ventricle to the left auricle. If the mitral valve is insufficient and its closure is incomplete, at every systole of the left ventricle a part of the blood is thrown back from it into the left auricle through the open space of the ostium venosum. This abnormal backward wave encounters the blood current coming in an opposite direction into the left auricle from the pulmonary veins. The meeting of these two opposing currents, as well as the stream of regurgitant blood pressing through the open chink in the mitral valves, sets the blood in a whirl, and this impinging upon the tense edges of the valves produces a loud, blowing, systolic murmur. We hear this murmur loudest at the apex of the heart, corresponding to the laws of conduction in the thorax; yet it usually is propagated so far that it may often be heard at the other cardiac orifices, although not so distinctly. A loud systolic mitral murmur can also be heard sometimes in the back, on the left, and occasionally on the right. In some few cases the systolic murmur of mitral insufficiency is heard best in the second left intercostal space. This is said to be due to the fact that the murmur occasioned by the commotion in the left ventricle is especially well conducted by the left auricular appendix which is near to the anterior chest wall (Naunyn). Curschmann has pointed out that this is particularly apt to happen in cases of incipient mitral insufficiency; but, as a rule, even in these cases, the murmur is found at the apex of the heart. In most instances the systolic muscle sound of the left ventricle, the so-called first sound of the heart, can also be heard besides the systolic murmur. It can be heard rather better if the ear is slightly removed from the ear piece of the stethoscope [meaning the monaural instrument]. Exceptionally the sound may be completely obscured by the murmur. The second sound is often not to be heard at the apex, probably because it is obscured by the relatively protracted murmur.

Since the left auricle, at each systole of the ventricle, receives blood from two sides—its normal quantity from the pulmonary veins, and, besides that, the abnormal blood wave from the left ventricle—it becomes much dilated. At the next diastole of the left ventricle the whole amount of blood collected in the auricle under increased pressure pours into the left ventricle through

the mitral valve, which is now wide open (supposing a pure insufficiency of the valve without any stenosis). We see, then, that in pure mitral insufficiency the left ventricle must be filled beyond the normal amount during the diastole. The left ventricle must also expel in the following systole an abnormally large amount of blood. Although by this contraction only a part of the blood reaches the aorta in the direction of the normal blood current while a part pours back into the auricle, the work of the left ventricle is, of course, excessive. Thus, in pure mitral insufficiency, the left ventricle is dilated as a result of its increased filling in diastole, and is hypertrophied as a result of its increased labor. The general arterial tension remains approximately normal. It is not increased, since a part of the abnormal amount of blood, which pours out of the left ventricle at every systole, flows backward into the auricle. So long as the left ventricle is completely emptied by vigorous contractions, the aorta receives about the normal amount of blood, and the radial pulse remains, therefore, in cases of pure mitral insufficiency, of about the normal strength and tension.

The anomalies in the movements of the blood in mitral insufficiency produce still other effects. We have already seen that the left auricle is dilated from its overfilling. It also becomes hypertrophied, so far as its weak muscular structure permits, but it is not in itself capable of compensating for the disturbance which the pulmonary circulation suffers from the mitral insufficiency, for the back current from the left ventricle, and the consequent high pressure in the left auricle, must plainly offer an abnormal hindrance to the flow of blood from the pulmonary veins. This stasis sets back through the pulmonary capillaries and arteries into the right ventricle. This may be recognized, on physical examination, by the change in the pulmonic second sound, which is louder, more valvular, and "accentuated," since the closure of the semilunar valves in the pulmonary artery now takes place under the abnormally high pressure which prevails in the arteries of the lungs. The right ventricle has the task of overcoming this abnormal stasis in the pulmonary circulation. It can overcome the abnormal resistance in the pulmonary circulation by increased work, and as a result it becomes hypertrophied. So long as the hypertrophy of the right ventricle suffices to maintain the normal pulmonary circulation, the stasis extends no farther backward, but in the later stages of heart disease we see the right ventricle becoming paralyzed, and more and more dilated as a result of stasis. The flow of venous blood from the body into the right auricle and ventricle is now rendered more difficult. The signs of venous stasis become manifest; the patient has a cyanotic hue, congestive œdema appears in the face and the extremities, symptoms of passive congestion of the liver, spleen, and kidneys appear, and, in short, there is developed the picture of an uncompensated heart disease.

If we now sum up the physical signs of mitral insufficiency, the different methods of investigation give the following results:

Inspection.—The cardiac region often seems rather prominent, as a result of the hypertrophy of the heart. This protrusion is most marked in young persons with a yielding thorax. The apex beat is, as a result of the dilatation and hypertrophy of the left ventricle, displaced toward the left and sometimes downward into the sixth intercostal space. It is more extensive and stronger than normal ("heaving"). The apex beat is somewhat displaced toward the

left as a result of the hypertrophy and dilatation of the left ventricle, and it is quite marked. Besides that, we often see a diffuse pulsation in the whole cardiac region. In the epigastrium we sometimes see an epigastric pulsation proceeding from the hypertrophied right ventricle. In cases which are no longer perfectly compensated the stasis in the veins of the body is rendered apparent by the general cyanotic appearance of the patient and the marked filling of the jugular veins in the neck. Undulatory or pulsating movements often occur in the latter (see tricuspid insufficiency, below).

Palpation.—By palpation likewise we perceive the abnormal vigor and extent of the apex beat, and the displacement of the same toward the left; and often, also, an extensive diffuse pulsation over the cardiac region, and in particular a distinct epigastric pulsation due to the right ventricle. We often feel a systolic thrill at the apex of the heart—a "cat's purr"—by laying the hand flat on the chest. The same whirl of blood, which is audible as a murmur, may be perceived as a fine tremor of the chest wall.

The radial pulse is quite strong and usually regular. The sphygmographic tracing of it shows nothing characteristic in mitral insufficiency.

Percussion.—This usually gives at first only a moderate increase of the heart's dullness to the left (*vide* illustration on page 384, showing the position of the different heart segments), but in the later stages there is at the same time an increase of the heart's dullness to the right, caused by hypertrophy and dilatation of the right ventricle. The whole area of cardiac dullness may finally extend two fingers' breadth beyond the right edge of the sternum, and to the left it may reach the mammillary line, or even pass far beyond it.

Auscultation.—At the apex of the heart we hear a loud, quite long, pure systolic blowing murmur, limited to the systole, either replacing the first sound or accompanying it. The second sound is often obscure or inaudible at the apex, but the pulmonic second sound is increased and accentuated. Auscultation of the vessels gives nothing characteristic.

2. **Stenosis of the Mitral Orifice (Mitral Stenosis).**—Mitral stenosis often develops in chronic endocarditis of the mitral valve, as a sequel to a previous insufficiency. The valve constantly grows stiffer and more rigid, and the

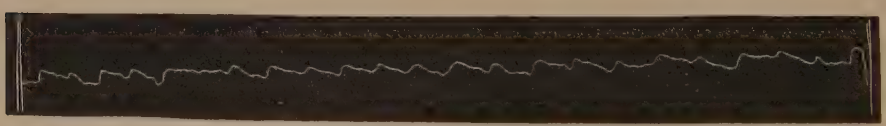


Fig. 60.—Pulse curve in marked mitral stenosis.

signs of stenosis gradually predominate over those of insufficiency. Hence we very often find stenosis and insufficiency of the mitral valve combined, but often the signs of stenosis are so much more prominent that we can properly speak of a pure mitral stenosis.

The disturbance which the circulation suffers in mitral stenosis is much greater than in mitral insufficiency. In mitral stenosis the orifice may finally become so narrow that it scarcely admits an ordinary lead pencil. The influx of blood into the left ventricle is accordingly much impeded. During the diastole of the left ventricle the blood must force its way through the stiff and narrow ring of the mitral valve. In this way, each time there are caused

irregular whirling movements in the blood, and abnormal vibrations of the mitral valve, giving rise in most cases to an audible diastolic murmur. In mitral stenosis the left ventricle receives less than its normal amount of blood, and therefore it has no direct occasion for hypertrophy, and in fact it is sometimes found at the autopsy to be comparatively small and to be crowded backward by the enormously dilated and hypertrophied right heart. If, nevertheless, we often find hypertrophy of the left ventricle in cases of mitral stenosis, it is for the reason that mitral stenosis usually develops gradually from a previous insufficiency of the valves. That is, the chronic endocarditis occasions, probably in every case, first, an insufficiency of the valve, which is later followed by stenosis as the change progresses. As insufficiency of the mitral valve leads to hypertrophy of the left ventricle (*vide supra*), we find it to persist even at a time when stenosis has become the prominent lesion. In other cases of stenosis, hypertrophy of the left ventricle is due to certain associated conditions, such as arteriosclerosis or chronic nephritis. And finally we should also consider Friedreich's theory, that extreme venous congestion may extend into the capillaries, and thence finally occasion abnormal resistance to the arterial circulation.

The radial pulse in mitral stenosis is approximately normal, as long as the ventricle is sufficiently filled with blood during diastole. In spite of the stenosis of the mitral valve, the left ventricle may be satisfactorily filled, and this is more likely if the action of the heart is slow, allowing a longer diastole; and if, also, the left auricle is still capable of vigorous contraction. If, however, the action of the heart is hurried, and the left ventricle is no longer sufficiently distended with blood during the diastole, the radial pulse becomes small and of low tension. Marked arrhythmia is very often present in mitral stenosis, probably because of the insufficient amount of arterial blood supplied to the myocardium and its ganglia, or because of a concomitant myocarditis.

The hindrance to the flow into the left ventricle in mitral stenosis soon leads to a marked stasis, which extends to the right side of the heart through the left auricle, and the pulmonary veins, capillaries, and arteries. The left auricle is dilated first (often to an enormous extent), and its walls are hypertrophied, but it can overcome only a very small part of the resistance at the mitral orifice. The right ventricle can, by more work, so increase the pressure in the pulmonary vessels that, in spite of the narrowed orifice, an approximately sufficient quantity of blood may pour into the left ventricle. Hence we find in mitral stenosis a very marked hypertrophy and dilatation of the right ventricle. The stasis in the pulmonary circulation, manifested objectively by the accentuation of the pulmonic second sound, has as a result a gradually developing ectasis of the pulmonary capillaries. Thickening of the intima of the pulmonary arteries and veins also usually develops. (See the chapter on Brown Induration of the Lungs.)

The results of physical examination are as follows:

Inspection.—The whole cardiac region may seem slightly prominent, as a result of the hypertrophy of the heart. This protuberance is most marked in children with their yielding thoracic walls. The heart's action is usually extended over a larger area, but in pure mitral stenosis the apex beat is no stronger than usual, though often displaced to the left. We frequently notice a marked pulsation in the epigastrium, produced by the right side of the

heart. The jugular veins are apt to be prominent, and show the different forms of undulatory and pulsating movement.

Palpation.—This also gives signs corresponding to the enlargement of the heart (sternal and epigastric pulsation). We sometimes feel the pulsation of the dilated right auricle (*vide infra*) even to the right of the sternum. In some cases we feel a diastolic thrill at the apex of the heart, which alone may almost establish the diagnosis of mitral stenosis. This thrill arises from the same vertiginous currents in the blood which form the basis of the diastolic murmur (*vide infra*). The radial pulse is small in every case of severe mitral stenosis, and is very often irregular.

Percussion.—Percussion shows, in the first place, an extension of cardiac dullness toward the right, reaching to the right edge of the sternum, or far beyond. Dullness also extends, as a rule, farther to the left than normal. This is in part due to the hypertrophy of the left ventricle (*vide supra*), in part to a dilatation of the right side of the heart, so great as to push the left ventricle backward and to the left. The great distention of the right ventricle causes an enlargement of the cardiac dullness upward.

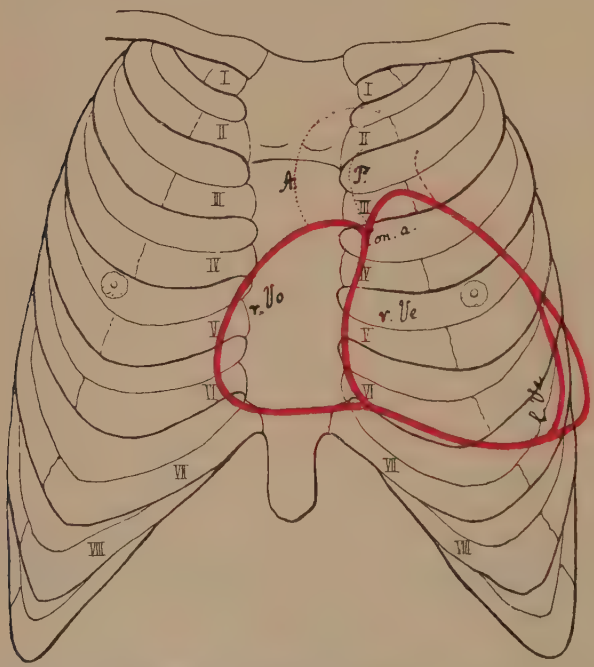


Fig. 61.—The position of the various cardiac segments in mitral stenosis. *r. Vo.* is the dilated right auricle; *r. Ve*, the dilated right ventricle; *Con. a*, the right conus arteriosus; *P.*, the pulmonary artery; *A*, the aorta; *l. Ve.*, the left ventricle pushed toward the left and backward.

We have often been able to convince ourselves by autopsies that the extension of cardiac dullness upward (absolute dullness beginning about the third rib), in mitral stenosis as well as in almost all other hypertrophies of the right ventricle, usually results from the right ventricle itself, and more especially from the dilatation of the right conus arteriosus. A marked extension of cardiac dullness toward the right and beyond the right sternal edge is almost without exception ascribable to dilatation of the right auricle. The right auricle, as is not always remembered, lies not above, but to the right of the right ventricle. In hearts with enlarged right ventricle, the right atrioventricular boundary almost always runs perpendicularly, so that the base of the heart is formed by the right auricle and the right ventricle. In the adjoining sketch (Fig. 61) the position of the various cardiac segments is demonstrated as found in almost all mitral lesions, especially

in mitral stenosis. The relations of the various portions to the enlarged cardiac dullness are easily recognized. Only exceptionally are the relations otherwise. Thus, we once saw a case of mitral stenosis in which the left auricle was so enormously dilated that it extended toward the right behind and beyond the right auricle, and was a prominent factor in the increase of cardiac dullness toward the right, observed during life. At times the upward increase in heart dullness is also caused by a distended right auricle.

Auscultation.—The characteristic auscultatory sign of mitral stenosis is the diastolic [presystolic] murmur at the apex. This is never so loud and blowing as the systolic murmur of insufficiency, but it usually sounds more rolling or rippling. It is loudest at the apex, and it is transmitted only slightly toward the base. Since, as has been said, the left ventricle in mitral stenosis is sometimes pushed to the left and backward by the very much enlarged right ventricle, in looking for the murmur we must often go far to the left, in order not to auscult the right ventricle only.

The origin of the murmur is easily explained. In the diastole of the left ventricle the blood current must force its way through the narrow mitral orifice, whence vertiginous movements arise in the blood, and produce the murmur. Since the blood flowing through the narrow orifice has a current of relatively slight intensity, the murmur produced by it cannot be very loud. Indeed, in the most extreme cases of mitral stenosis, the murmur is particularly apt to be very faint, and when the heart's action is hurried and irregular, entirely inaudible. Not infrequently the murmur is not heard until the second half of diastole, that is, when the contraction of the left auricle gives a fresh impulse to the current of blood streaming through the stenosed orifice. A murmur of this sort, which is audible only at the end of diastole, and passes with a distinct crescendo directly into the loud systolic sound and terminates with this, is called a presystolic murmur. This murmur can often be plainly felt if the hand is laid upon the apex of the heart, as a presystolic thrill.

It is not very exceptional to find no murmur audible in cases of extreme mitral stenosis. If such cases do not come under observation till the last stage of the disease, the mitral stenosis may readily be overlooked. We have ourselves repeatedly found, in cases of mitral stenosis, that as the lesion grew worse the distinct diastolic or presystolic murmur gradually and completely disappeared. This is explained by the fact that as the chink in the valve grows narrower and the weakness of the heart greater, the blood is not forced through the narrow orifice with sufficient vigor to cause audible vibrations of the thickened valves. If the left ventricle is wholly displaced backward by the enormous increase in size of the right ventricle, we have also less favorable conditions for the propagation of the waves of sound from the mitral valve to the ear.

The first sound at the apex is maintained in pure mitral stenosis. Indeed, it is often noticeably loud and valvular. All the later observations confirm the view that the systolic sound is a muscle sound, and consequently it is probable that this vigorous first sound is due to the contraction of the left ventricle, which is often hypertrophied (*vide supra,* page 383), but nevertheless, as a result of the stenosis, only imperfectly filled. The strength of the first ventricular sound in mitral stenosis affords, at any rate, a marked

contrast to its weakness in aortic insufficiency (*vide infra*). We have a strong sound when the ventricle is little filled, diminution and dullness of sound when the ventricle is overfilled. If insufficiency of the valve coexists, we hear a systolic murmur usually along with the first sound. In mitral stenosis there is often a very marked accentuation of the pulmonic second sound, the result of the abnormally high tension in the pulmonary artery. The pulmonic second sound is, however, very often only slightly accentuated without our being able always to assign a real cause therefor. Perhaps there is a difference in the vibratory power of the valves. In anæmic, sickly individuals, and also in cases of simultaneous insufficiency of the tricuspid valve (*vide infra*), there is no accentuation of the second pulmonic sound. Sometimes, as a result of extensive congestion in the systemic veins, the pressure in the pulmonary artery, and with it the accentuation of the second pulmonic sound, appear to lessen. Very often the second sound at the base of the heart is "split" (reduplicated). The closure of the semilunar valves in diastole does not happen at the same time in the pulmonary artery and in the aorta, on account of the unequal tension in the two vessels, so that consequently the two sounds are heard, one shortly after the other. But other, still unknown, factors may operate. At times we may hear no murmur in mitral stenosis (*vide supra*), but only a loud valvular systolic and diastolic sound.

Mitral stenosis is one of the severest forms of heart disease. It almost always causes greater subjective disturbance than mitral insufficiency. Hypertrophy of the right ventricle may, indeed, maintain for a time an approximately complete compensation, but the signs of marked stasis in the pulmonary circulation, and further, in the veins of the body, are apt to appear quite early. The disturbances of compensation which occur in mitral stenosis are, indeed, particularly susceptible of treatment, so that for many years there may be times of improvement, alternating with times of general circulatory disturbance and marked subjective symptoms. But at last it becomes impossible to regulate the circulation. The dyspnœa grows worse, and finally death occurs, usually ushered in by increasing dropsy.

3. **Insufficiency of the Semilunar Valves of the Aorta.**—Insufficiency of the aortic valves is due most frequently to contraction of the free edges of the valves. Tears, perforations, or adhesions of the valve to the wall of the vessel more rarely lead to insufficiency. The cause of all these changes is either a valvular endocarditis, which is usually a sequel of articular rheumatism, or a general arterial atheroma, which extends gradually from the intima of the aorta to the valves. We have already referred to syphilis as a not very infrequent cause of aortic lesions. A question of practical importance is whether violent bodily exertion may suddenly occasion the partial laceration of an aortic valve. Many clinical observations, including a recent case of the author's, seem to show the possibility of this extremely rare occurrence.

The function of the aortic valves is to close tightly at the time of diastole of the left ventricle, in order to prevent any return of blood from the aorta into the ventricle. If these valves are insufficient—that is, if they do not close perfectly at each diastole—there is a return current of blood from the aorta into the left ventricle.

This regurgitant diastolic wave sets the tense edges of the valve in vibration as it passes over them. Furthermore, the two currents of blood impinging upon each other in the left ventricle, the one an abnormal regurgitation from the aorta, the other the normal stream from the left auricle, produce irregular whirling motions in the blood. All these vibrations are propagated to the surrounding structures, and produce the long-continued, blowing, diastolic, and remarkably characteristic murmur of aortic insufficiency.

Aortic insufficiency causes an immediate and great increase in the demands upon the left ventricle, because of its abnormal distention, for it receives, as we have already said, not merely its normal quantum of blood from the left ventricle, but also the blood which regurgitates through the leaking valves of the aorta. It is consequently overfilled at every diastole, and finally becomes permanently distended. Dilatation of the left ventricle forms a constant anatomical lesion in every case of aortic insufficiency, and is shown not only in the dilatation of the whole ventricular cavity, but also in the very characteristic flattening of the trabeculæ and of the papillary muscles. There is usually some fibrous thickening of the endocardium at the spot upon which the regurgitant blood current is continually impinging. The left ventricle possesses sufficient reserve strength to discharge its contents completely for a long period by means of increased effort. This is indeed a task like that of Sisyphus, since the portion of the blood which is thrown into the aorta is constantly rolling back into the ventricle. The increased demands, however, lead necessarily at last to a hypertrophy of the left ventricle, often greater than is seen under any other conditions.

From the facts enumerated we can easily understand the physical signs of insufficiency of the aortic valves.

Inspection.—The great dilatation of the left ventricle often causes a marked protrusion of the whole cardiac region. The diffuse and very strong apex beat, displaced downward and to the left, is especially striking. It may usually be seen in the sixth intercostal space, outside the left mammillary line, and sometimes even at the anterior axillary line. Besides, we often see a marked diffuse tremor of the whole cardiac region. There is marked pulsation of the carotid arteries in the neck. The jugular veins show undulation and pulsation, when at last compensation begins to fail.

Palpation.—We can appreciate the heart's action to a still greater extent by palpation than by inspection. The apex beat is very resistant, massive, and plainly heaving—that is, the finger or stethoscope applied to the apex is lifted by the beat at every systole. In rare cases a diastolic thrill, corresponding to the diastolic murmur, can be felt over the base of the heart. In two such cases observed by us the murmur had a marked musical character (*vide infra*). The appearances in the arteries are given below.

Percussion.—Percussion gives an extension of the cardiac dullness to the left, beyond the left mammillary line and even to the anterior axillary line, caused by the hypertrophy and dilatation of the left ventricle. The upper boundary of the cardiac dullness is normal, or it may extend up to the third rib. The right boundary is in its normal place at the left border of the sternum, but it may also be pushed farther to the right, either because the large left ventricle of itself causes an extension of the whole heart to the right, or because the right ventricle is also hypertrophied. The latter change occurs

in pure aortic insufficiency when the compensation is no longer complete, and the stasis extends backward from the left ventricle, through the pulmonary circulation, into the right side of the heart.

It may also be remarked here that, in insufficiency of the aortic valves, the ascending aorta is often considerably dilated by the marked impulse of the volume of blood pouring into it. A moderate degree of dullness is found over the dilated aorta, which may sometimes be made out at the sternal end of the second right intercostal space.

Auscultation.—Insufficiency of the aortic valves is characterized by a long-drawn, loud, blowing, diastolic murmur, the origin of which has been explained above. The place in which the murmur is heard loudest is not the sternal end of the second right intercostal space, the ordinary point for auscultation of the aorta, but it almost always lies farther to the left. Corresponding to the backward current of blood toward the left ventricle, which begets the murmur, we hear the latter loudest over the upper part of the sternum or even at its left border. In some cases the murmur assumes a marked "musical character"—that is, there is a definite high musical tone, which is due to a tendinous fiber arising from a wearing away of the valve, and set in vibration by the diastole, or to some similar cause. The diastolic murmur is often audible at the apex, but it is faint there. It is only in a few exceptional cases that there is no diastolic murmur in aortic insufficiency. Sometimes we hear not only the murmur, but also the diastolic sound of the closing valve. During systole we only rarely hear over the aorta a pure, loud, first sound, but almost always a short systolic murmur. This murmur may, of course, be due to accompanying stenosis of the aortic valves, but yet is frequent when there is insufficiency alone. It is explained by O. Rosenbach as being due to the fact that at the beginning of the systole of the left ventricle, the diastolic flow of blood has not yet completely ceased, so that the emerging blood stream encounters this opposing current. This meeting of the two blood currents in the root of the aorta during systole causes the vibrations which give rise to the short systolic murmur. It is to be noted that this circumstance also may perhaps have some influence upon the development of the hypertrophy of the left ventricle.

It is very interesting and important that, as Traube pointed out, we find the first sound at the apex scarcely ever loud and pure, but often very indistinct and muffled; or else we hear a short systolic murmur instead of it. This veiling of the first sound at the apex of the heart has theoretic interest, because it has been employed as an argument against the view that the first mitral sound is a muscular sound; for it is not, in fact, at once apparent why the hypertrophied left ventricle should so often fail to produce a distinctly audible tone by its contraction. But, as we have already pointed out (see page 387), the probable explanation lies in the previous overdistention of the left ventricle during diastole. This renders the systolic contraction difficult and somewhat slow, and may be the cause of the indistinctness of the muscle sound. In later stages of the disease we may also adduce parenchymatous degeneration of the myocardium as an explanation of its feebleness. The systolic murmur, often heard at the apex in aortic insufficiency, may depend upon a coexisting true mitral insufficiency, but it is probably often due to a relative insufficiency of the mitral, since the valves, which are normal in

themselves, can no longer cause a perfect closure of the left mitral orifice now that the left ventricle is dilated.

Symptoms in the Peripheral Arteries.—Such remarkable symptoms are found in the peripheral arteries in aortic insufficiency that they demand a brief special description. The first striking symptom is the strong pulsation not only of the larger but also of the smaller arteries, even those the pulsation of which is not generally visible. We see and feel not only a strong pulsation in the carotids, but also in the tortuous brachial artery, in the radial, ulnar, temporal, dorsalis pedis, etc. We sometimes feel an arterial pulse in the liver through the abdominal walls.

The rapid decline of the pulse—the *pulsus celer* [Corrigan's pulse]—is most characteristic of aortic insufficiency, and is to be felt especially in the radial artery, but also in the femoral, dorsalis pedis, and other vessels. An abnormally large quantity of blood is thrown into the arteries from the hypertrophied and dilated left ventricle; hence the high ascent of the pulse; but since the distended artery quickly contracts again, and particularly as at the next diastole of the ventricle the blood escapes in two directions, into the capillaries and back into the ventricle, an abnormally rapid and deep decline of the pulse follows the high ascent of its wave—a condition which explains the "jumping," "springing" pulse (*pulsus celer*) of aortic insufficiency. The quality of the pulse may be plainly recognized also in its sphygmographic tracing (see Fig. 62). The abnormal backward wave may even be detected in the capillaries. We often see a marked pallor of the finger nails, or of the skin of the forehead when reddened by rubbing, at every diastole of the heart in patients with aortic insufficiency—Quincke's capillary pulse.

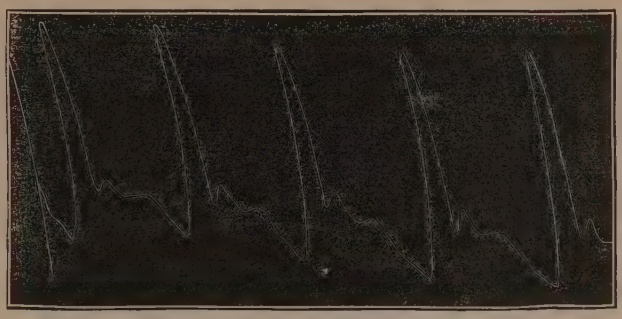


Fig. 62.—Pulse curve in aortic insufficiency.

The auscultatory phenomena over the arteries are connected partly with the changing conditions of tension of the arterial walls. We very often hear a short, rough, systolic murmur in the carotid. The second sound, which is well known to be the transmitted aortic second sound, is absent. Instead of it we sometimes hear faintly transmitted the aortic diastolic murmur. The sound of the medium-sized and smaller arteries is very characteristic. By applying the stethoscope lightly we hear over the femoral, the brachial, and often over the radial, the ulnar, the palmar arch, and the dorsalis pedis, a marked valvular sound, which is changed by pressure on the artery, especially in the larger arteries, to a loud stenotic murmur. The quicker the pulse, the more certain are we to hear these sounds in the arteries. In the most marked cases of Corrigan's pulse these vascular sounds are so loud that we may hear almost anywhere below the knee, by means of a stethoscope, a valvular sound. The double sound in the femoral (Traube's double sound) is quite a frequent

phenomenon, about the origin and significance of which there has been much discussion. The double sounds either follow each other shortly, so that the first seems something like a preparatory blow for the second, or they are separated from each other by a longer interval, like the two sounds of the heart. Traube explained the origin of the first sound by the sudden tension of the vessel wall, as in the simple femoral sound, and the second sound by the sudden relaxation of it. Friedreich has pointed out in regard to this that, in coexisting tricuspid insufficiency, a sound may also be produced in the femoral vein by tension of the venous valves. The double sound in the femoral may probably have different causes of origin. It is, of course, by far the most frequent in aortic insufficiency, but it has also been repeatedly observed in other forms of heart disease, as in mitral stenosis. The so-called Duroziez's double murmur in the femoral is more rare, and it is noticed almost exclusively in aortic insufficiency. By pressing the stethoscope on the femoral, we hear two murmurs plainly distinct from each other, of which the first comes from the passage of the systolic blood wave, and the second from the passage of the abnormal backward wave coming from the periphery of the vascular system through the artificially contracted vessel.

While the well-marked Corrigan's pulse and the arterial sounds associated with it are so characteristic, they appear with great distinctness only in some cases of aortic insufficiency; while in other and often apparently similar cases they are indistinct or quite absent. Probably this difference depends at least in part upon a difference in the elasticity of the arterial walls. At any rate, we have seen well-marked Corrigan's pulse and sounding arteries in youthful patients; while in elderly persons with accompanying arteriosclerosis or similar changes, these phenomena are not apt to be striking.

Aortic insufficiency is a comparatively favorable form of heart disease, since it may be almost perfectly compensated for years by hypertrophy of the left ventricle. Many patients with moderate aortic insufficiency feel perfectly well, and are even capable of quite hard work. They have not the slightly cyanotic hue which almost all patients with mitral disease exhibit, but they have a normal or even a pale complexion. If, however, the signs of disturbed compensation once appear in aortic insufficiency the severest sequelæ may develop quite rapidly. In aortic insufficiency it is exceptional to see such repeated changes from bad to good, and good to bad, as are often observed, for instance, in mitral stenosis. If the left ventricle becomes enfeebled, it can no longer satisfy the excessive demands made upon it. Passive congestion ensues, extending backward through the pulmonary circuit into the systemic veins, even while the pulse may still seem to be powerful. The average arterial tension becomes subnormal, dyspnœa increases, and there are attacks of cardiac asthma. Œdema appears, and the patient dies with the symptoms of anasarca. We will speak more fully below of certain intercurrent events in aortic insufficiency, such as cerebral hemorrhage and pericarditis.

4. **Stenosis of the Aortic Orifice.**—Except for the mild forms of aortic stenosis, which often come on with aortic insufficiency, aortic stenosis is a rare disease. It arises from marked thickenings and calcifications, and especially from adhesions of the aortic valves to one another. The stenosis may become so considerable that the orifice is finally reduced to a mere fissure, through

which the left ventricle must force the blood at its systole. The fluttering of the valves and the vertiginous movements in the blood thus arising produce a loud systolic murmur. The left ventricle is compelled to do greater work in consequence of the increased resistance of the aortic orifice, and hence becomes hypertrophied. In spite of the increased effort, however, comparatively little blood reaches the arterial system, and consequently the radial pulse is small and the arteries contracted.

Inspection and Palpation.—Upon physical examination of the heart we find, in the first place, the apex beat displaced outward as a result of the hypertrophy of the left ventricle, and also often more powerful than normal. It may, however, be noticeably feeble, perhaps because of the slowness of the systole. A former explanation of this feebleness was the diminution of the recoil of the apex (the Gutbrod-Skoda theory of the apex beat).

Percussion.—Percussion gives an extension of the heart's dullness to the left. The right ventricle is also dilated and hypertrophied to a moderate degree in the later stages, if the stasis extends backward through the pulmonary circulation.

Auscultation.—On auscultation, we hear over the aorta a very loud "sawing," long-drawn, systolic murmur, which is usually transmitted to the right, corresponding to the course of the aorta, in distinction from the diastolic murmur of aortic insufficiency. It is usually to be heard loudest at the sternal end of the second right intercostal space, but it is audible to a lesser extent over almost the whole heart. It is usually quite loud over the carotids. The systolic sound at the apex is apt to be feeble. The second aortic sound is likewise faint or even inaudible. If there is coexisting insufficiency of the valve, the second aortic sound is replaced by a diastolic murmur.

The pulse has been already described. It is small, and often surprises one by its contrast with the strength of the apex beat. In well-compensated cases it is regular, and often moderately or even extremely slow. This slow pulse of aortic stenosis is often explained as a compensatory change in the heart's action, appropriate to the existing lesion—the systole being lengthened, an increased amount of blood can be driven through the narrow aortic orifice. But the slowing of the cardiac action is really, in main part, a prolongation of the diastole, and therefore the slow pulse is probably due chiefly to the fact that the wall of the left ventricle is ill supplied with blood, just as in the case of sclerosis of the coronary arteries. The sphygmographic tracing of the radial pulse (see Fig. 63) shows a low wave and a comparatively slow rise and fall.

Fig. 63.—Pulse curve in stenosis of the aortic orifice.

Aortic stenosis of slight or moderate degree may be tolerably well borne by the patient. We have even seen a man with well-marked aortic stenosis who for years did not have the slightest subjective symptoms of heart disease, until he finally died with an acute recurrent endocarditis. When the stenosis is more complete we sometimes have a most peculiar clinical picture. The pulse is very infrequent, as low as thirty to twenty-four beats in a minute. From time to time there are attacks of vertigo or syncope, the patient often

falls, and has epileptiform attacks. These seizures, which may be repeated for months or even for some years, are probably connected with a sudden anæmia of the heart and brain. We have observed this remarkable group of symptoms particularly in elderly persons with aortic stenosis, due to arteriosclerosis (*vide infra,* page 417). In other respects the course of aortic stenosis is similar to that of the acute valvular diseases, and in the same way terminates in general circulatory derangement with its results.

5. **Insufficiency of the Tricuspid Valve.**—Insufficiency of the tricuspid valve is extremely rare as an independent disease of the heart, but a secondary insufficiency of the tricuspid is quite frequent, and is therefore of practical interest, as it complicates other already existing valvular diseases in the left side of the heart. It arises either from a secondary endocarditis, affecting the tricuspid, in quite an analogous manner with mitral insufficiency, or it is a so-called relative insufficiency. This name we give to that form of insufficiency which develops when the edges of the tricuspid valve, normal in themselves, at last fail to meet one another, from the increasing dilatation of the right ventricle, or at any rate from the inability of the enfeebled right ventricle properly to close the valve.

The necessary result of tricuspid insufficiency is, that in every systole of the right ventricle a backward current passes through the open tricuspid orifice into the right auricle, and thence into the veins of the body. The tricuspid insufficiency ensuing in other forms of heart disease must therefore increase the stasis in the veins of the body, and it is thus far an unfavorable complication. It has a compensatory significance only as it affords relief to the pulmonary circulation. Since a part of the blood passes back from the right ventricle into the veins, less blood than usual must reach the pulmonary arteries. The decrease in tension thus produced in these arteries makes itself apparent on auscultation, since the accentuation of the pulmonic second sound in valvular disease of the mitral orifice diminishes when tricuspid insufficiency takes place.

That tricuspid insufficiency must result in a hypertrophy of the right ventricle is explained in just the same way as the hypertrophy of the left ventricle in mitral insufficiency, from the increased influx of blood at increased tension into the right ventricle during diastole; but this effect of tricuspid insufficiency can rarely be made out in any individual case, since the right ventricle is usually already hypertrophied as a result of the disease in the left side of the heart.

The most important symptom from which we can diagnosticate tricuspid insufficiency is the venous pulse. The cause of this is the backward wave of blood produced at each systole of the right ventricle. So long as the venous valve above the bulbus jugularis is closed, we usually see only a "bulbar pulse," but very soon this valve also yields to the continued impulse of the blood, and then a strong, purely venous pulse is visible along the whole course of the jugular vein up to the vicinity of the mastoid process. The contraction of the right auricle very often causes a decidedly weaker elevation of the vein, which just precedes the marked pulsation caused by the ventricular systole (anadicrotic venous pulse). On account of the straighter course of the right innominate vein, the jugular venous pulse is often stronger on the right side than on the left. We must state, however, that the jugular venous pulse is

not an absolutely certain sign of tricuspid insufficiency, since it may arise in hypertrophy of the right side of the heart without any insufficiency of the tricuspid from the closure of the valves.

If there is pulsation in the bulb of the jugular vein and the jugular valve is still capable of closing, a low, audible, venous, valvular sound may be produced by its closure. A sound may also arise in tricuspid insufficiency, as has been already said, from the tension of the valves in the femoral vein. A visible pulsation in the larger veins of the extremities is very rare, but in tricuspid insufficiency we quite frequently feel a venous pulsation in the liver, which organ is usually enlarged by passive congestion. This may be quite apparent even in many cases in which the jugular venous pulse is absent, because the veins in the liver are without valves. [It is best appreciated by means of bimanual palpation.]

Auscultation over the right side of the heart gives a systolic murmur in insufficiency of the tricuspid, arising from the regurgitating blood current. This may be heard loudest over the lower part of the sternum, or at the sternal end of the right fifth rib. The significance of this murmur in diagnosis, however, is impaired by the fact that it cannot always be separated from the systolic mitral murmur that often coexists.

6. **Stenosis of the Tricuspid Orifice.**—Stenosis of the tricuspid orifice is an extremely rare disease, and hence it is without practical significance. It has usually been observed, up to the present time, as a congenital form of heart disease, almost always combined with other anomalies of development in the heart.

The physical signs of tricuspid stenosis can easily be constructed theoretically. The first result must be a marked dilatation of the right auricle, and the occurrence of a diastolic or presystolic murmur over the right side of the heart. From the rarity and complex character of the cases, however, we have so far seldom had an opportunity to confirm these theories at the bedside.

The prognosis of this form of heart disease is very unfavorable, since a long-continued compensation by increased labor on the part of the right auricle is scarcely conceivable.

[Seventy cases of tricuspid stenosis have been collected by Bedford Fenwick, whose analysis affords good grounds for thinking that the lesion is often acquired. In fifty per cent of the cases there was a clear history of rheumatism, and nearly all of the patients were more than twenty years of age at the time of death.

This lesion is rarely found alone, being almost invariably combined with mitral stenosis; all but eight of the cases were in women. Fenwick thinks that the influence of sex lies in the less onerous nature of the work of women than of men, the granulating edges of the valves being kept more in apposition, thus healing with adhesion and causing obstructions at the orifice.]

7. **Insufficiency of the Pulmonary Valve.**—Insufficiency of the pulmonary valve is also a very rare form of heart disease. It occurs as a congenital anomaly, often combined with other failures of development, or as a disease acquired after birth. The anatomical changes in the valves, which lead to insufficiency, are precisely analogous to those which cause insufficiency of the aortic valve.

The physical signs of this form of valvular disease consist chiefly of a

marked dilatation and hypertrophy of the right ventricle, to be made out by percussion, and of a loud diastolic murmur over the pulmonary valve. These signs are explained in just the same way as the precisely analogous signs in the left ventricle in aortic insufficiency.

In general, pulmonary insufficiency, like aortic insufficiency, seems to be compensated quite well for a long time by hypertrophy of the right ventricle. In many cases a coexisting patency of the foramen ovale also seems to be of favorable influence, as it lessens the stasis in the right auricle and the veins of the body, while it renders easier the filling of the left ventricle.

8. **Stenosis of the Pulmonary Orifice (Pulmonary Stenosis) and the other Congenital Diseases of the Heart.**—(1) Congenital Pulmonary Stenosis.—While the stenosis of the pulmonary orifice acquired in later life is so rare that it has only slight practical significance, the congenital pulmonary stenosis is of far greater importance. It is, on the whole, the most frequent of the congenital forms of heart disease. Its origin is to be referred either to an endocarditis of the pulmonary valves during fetal life, or to anomalies in the development of the heart. The stenosis is often situated not merely at the pulmonary orifice itself, but farther back in the conus arteriosus, which seems to be narrowed by the formation of myocardial cicatrices. The pulmonary artery is often also narrowed as a whole. In the majority of cases we find, in addition, other anomalies of development in the heart, especially patency of the foramen ovale, great defects in the ventricular septum, and in about half the cases, patency of the ductus Botalli, etc.

The symptoms of congenital pulmonary stenosis sometimes appear soon after the birth of the child. The first thing that strikes us is the marked cyanosis, which is constant, or else comes on with crying, or with movements of the body. Many children, however, reach a fair age, five or ten years, or even fifteen. In some cases the heart disease may be so perfectly compensated that the child may be comparatively well for a time, and severe disturbances may not appear for several years.

As a rule, children with congenital pulmonary stenosis present a very striking appearance. The cyanosis is especially noticeable in the face, the lips, the nose, and the hands and nails. The parts mentioned feel cool. The eyes are often somewhat prominent, and there is a slight œdematous swelling about them. The peculiar club-like thickening of the terminal phalanges of the fingers and toes, a result of stasis, as seen also in many cases of bronchiectasis, is very characteristic. The nails also present a characteristic claw-like curvature.

The whole development of the child is remarkably retarded. It often seems several years younger than it is. The muscles and fatty layer are slight. The gums are sometimes very spongy and disposed to bleed. In severe cases the child suffers from faintness, vertigo, etc.

On physical examination of the heart, we usually find the cardiac region rather prominent. Percussion gives an increase of the heart's dullness, especially toward the right. This extension of the dullness is due to the hypertrophy of the right ventricle, which must arise in the same way as hypertrophy of the left ventricle in aortic stenosis. On auscultation, we hear a loud systolic murmur, which is perceptible over the whole heart, but which has its greatest intensity at the sternal end of the second left intercostal space. The

eddies of blood, which produce the murmur, may also often be felt by the hand as a systolic thrill. In some cases, however, we miss the murmur in pulmonary stenosis, just as in mitral stenosis. The pulmonic second sound is weak or inaudible, or it is replaced by a murmur if there is also insufficiency of the valves.

The course of congenital pulmonary stenosis is always unfavorable. As has been implied, few children get beyond the age of fifteen years. Death ensues, either with general disturbances of compensation evidenced by dyspnœa and dropsy, as in every other form of heart disease, or from complications. Among the latter, we may mention especially phthisis, which, probably because of the deficient blood supply, strikingly often develops in children with congenital pulmonary stenosis.

(2) The Remaining Congenital Lesions of the Heart.—Inasmuch as other congenital lesions than pulmonary stenosis have but slight clinical importance, we will limit ourselves here to a brief review of the same.[1]

(*a*) Patency of the foramen ovale is a comparatively frequent lesion, whether alone or combined with others. Physical signs are mostly absent. In a few cases a presystolic murmur has been heard. When mitral insufficiency coexists with a patent foramen ovale, venous pulsation may be caused.

(*b*) Defects in the septum between the ventricles. These are most frequently situated in the posterior section of the anterior septum, and they likewise are associated with other anomalies, such as abnormal distribution of the arteries, pulmonary stenosis, or defects in the septum between the auricles. Sometimes the patency of the septum gives rise to a systolic murmur, but a diagnosis of the condition during life is scarcely ever possible.

(*c*) Persistence of the ductus Botalli. Inasmuch as this contributes blood from the aorta to the pulmonary circulation the pressure in the latter is raised, hence there is to be observed accentuation of the second pulmonic sound and hypertrophy of the right ventricle. There is sometimes also a loud systolic murmur.

(*d*) We have already spoken of congenital stenosis of the tricuspid valve. Congenital narrowing of the mitral valve and of the aortic valves may also occur, but they are extremely rare.

9. **Combined Valvular Diseases of the Heart.**—Although in what has preceded we have dealt with the several forms of valvular disease of the heart separately, in order to present them in a general way, yet in reality combinations of them often occur in great variety. We find especially, as has already been mentioned, stenosis of an orifice coexisting with insufficiency of the accompanying valve; but diseases of two or more different valves are not infrequent, combined in the most diverse manners. The physical signs of these "combined forms of heart disease" may, of course, be inferred from the signs of anomalies of single valves, but the phenomena are often so complicated that the diagnosis of combined heart disease is generally much harder than that of the simple forms. Sometimes the single forms neutralize one another in their action. For example, the left ventricle is usually small in pure mitral stenosis, but, if aortic insufficiency be also present, it is neverthe-

[1] A more extensive presentation of the subject can be found in the article by Rauchfuss in Gerhardt's "Handbuch der Kinderkrankheiten," vol. iv, and in text-books on pathological anatomy —for example, Orth's.

General Comparison of the Most Important Physical Signs in Valvular Disease of the Heart

Form of Heart Disease.	Inspection.	Palpation.	Percussion.	Auscultation.
1. *Mitral insufficiency.*	Strong apex beat, often somewhat displaced outward.	Systolic thrill at the apex. Quite strong radial pulse.	Hypertrophy of the left, later of the right, ventricle.	Loud systolic murmur at the apex. Pulmonic second sound accentuated.
2. *Mitral stenosis.*	Area of cardiac impulse enlarged, epigastric pulsation.	Diastolic thrill at the apex. Small and often irregular pulse.	Hypertrophy of the right ventricle.	Diastolic or presystolic murmur at the apex. First sound sometimes loud. Pulmonic second sound accentuated, and sometimes double.
3. *Aortic insufficiency.*	Apex beat very strong, displaced downward and to the left. Visible pulsation of the medium-sized and smaller arteries.	Very strong, heaving apex beat. *Pulsus celer.*	Marked hypertrophy of the left ventricle.	Loud diastolic aortic murmur, especially over the upper part of the sternum. Sounds in the arteries (femoral and brachial sounds, etc.). Sometimes a double sound or double murmur in the femoral.
4. *Aortic stenosis.*	Apex beat displaced to the left.	Heart's action not very strong. Pulse small, infrequent, sometimes slow.	Hypertrophy of the left ventricle.	Loud systolic aortic murmur, transmitted to the right.

less found dilated, at least to a certain degree. The influence of an absolute or relative tricuspid insufficiency on the action of mitral disease, especially the decrease in tension in the pulmonary vessels caused by it, and also the diminished accentuation of the pulmonic sound, have been mentioned above.

In reference to the clinical symptoms of combined heart disease we may say, on the whole, that, in a large number of cases, the disease of *one* valve stands out as predominant in the whole picture of the disease. The other anomalies are only slightly noticeable, and often of later date. Hence, we may find at the autopsies of patients, who during life have shown the symptoms of disease of only one particular valve, unimportant changes on the other valves, which have been without clinical significance.

[Bramwell reports that of 131 cases with macroscopic valvular lesion, the tricuspid was implicated in 33.58 per cent; in all but 12 per cent of these the changes were recent. Hence he thinks that tricuspid endocarditis is generally recovered from, and this he attributes to the relatively small strain to which that valve is subjected. The obvious therapeutic deduction is the importance of rest in mitral endocarditis.]

GENERAL SEQUELÆ AND COMPLICATIONS OF VALVULAR DISEASE OF THE HEART—SUBJECTIVE SYMPTOMS —SEQUELÆ IN THE HEART ITSELF

After having discussed, in what precedes, the mechanism of the single forms of valvular disease, and the physical signs derived from it, we must now describe a number of symptoms and sequelæ which may be present to a greater

or less degree in all forms of valvular disease. With them we must also mention certain peculiarities of the individual forms.

1. **Subjective Symptoms.**—Fully compensated heart disease may exist, at least for a long time, without any subjective symptoms. This is especially the case in aortic insufficiency, more rarely in mitral insufficiency. Stenosis of the mitral or of the aortic valves almost always causes subjective symptoms. These symptoms often do not exist so long as the patient keeps perfectly quiet physically and mentally, but they develop under appropriate circumstances, especially upon bodily exertion.

The existing subjective symptoms in heart disease are by no means always referred, in the first place, to the heart itself. It sometimes happens that the patient comes to the physician complaining of various digestive disturbances, or in other cases of headache, vertigo, etc. The physical examination alone leads us to recognize the heart disease. As a rule, the patient's first and chief complaint is directed toward his difficulty in breathing. The shortness of breath, which increases on any physical exertion, comes on quite early in many cases. In the later stages it is almost always the most distressing symptom. The causes of dyspnœa in heart disease are manifold. In the first place dyspnœa results from congestion of the pulmonary circuit, with consequent slowing of the circulation in the lungs, and limitation of the exchange of gases in those organs. In later stages the anatomical changes in the lungs contribute also to an increase of the dyspnœa (*vide supra,* the chapter on Brown Induration of the Lungs). Basch ascribes special importance to the fact that the distended capillaries encroach upon the alveoli as a result of the passive congestion. This broadens the alveoli ("swelling of the lungs"), but at the same time impairs the mobility of the lungs ("rigidity of the lungs"), and thus embarrasses the exchange of gases in the lungs, just as in emphysema. The occurrence of the "rigidity of the lungs" is to be admitted; but I cannot admit a "swelling of the lungs" according to Basch's ideas. In microscopic preparations of lungs affected by passive congestion I find the alveolar spaces diminished rather than enlarged.

The secondary bronchitis of heart disease is a very great factor in the dyspnœa. This bronchitis is a frequent result of the pulmonary stasis. Often the respiratory distress increases and decreases simultaneously with corresponding variations in the bronchitis. A purely mechanical cause of dyspnœa is the compression of the lower portion of the left lung by great cardiac hypertrophy. The highest grade of dyspnœa is reached when finally hydrothorax, hydropericardium, and pulmonary œdema are developed. From what has already been stated, it is easy to see why lesions of the mitral valve which directly impair the pulmonary circulation lead sooner to shortness of breath than do lesions of the aortic valves. Finally, it is self-evident that the condition of the heart has the greatest influence upon the degree of dyspnœa present, for all the phenomena of pulmonary congestion must increase or diminish, according to the functional integrity of the heart, and particularly of the left ventricle. If the contractile power of the left ventricle grows less, dyspnœa must at once increase. Not infrequently conditions of cardiac weakness develop with considerable suddenness, and occasion attacks of dyspnœa which are termed cardiac asthma. At the same time the enfeeblement of the left ventricle often leads to a transient stasis of the pulmonary vessels, and, as a consequence thereof,

to a congestive transudation into the bronchi. The patients feel impelled to cough and expectorate small or even large quantities of a serous or sero-sanguineous sputum.

Palpitation is the first subjective symptom to be mentioned of those which are referred directly to the heart. It is not yet accurately determined under what circumstances the action of the heart is perceived by the patient himself. We sometimes see an uncommonly strong action of the heart, as in aortic insufficiency, which is not perceived at all subjectively. In other cases, where objectively the heart is not especially active, palpitation forms the patient's chief complaint. It usually first appears when the heart disease ceases to be fully compensated. It is increased or first excited by physical exertion or mental excitement. In many patients attacks of palpitation occur without any discoverable external cause, due apparently to nervous disturbance. They are sometimes associated with a striking acceleration of the pulse, the so-called tachycardia.

Pain in the cardiac region is only rarely present in heart disease. The patients more frequently complain of an indefinite feeling of pressure and oppression in the chest, but still there do occur, particularly in aortic insufficiency, attacks of violent pain in the front part of the chest and the region of the heart, radiating to the shoulders and arms, and associated with a general feeling of extreme anxiety and weakness. Such conditions are termed angina pectoris, or attacks of stenocardia. They are probably generally dependent upon the coexisting aortic sclerosis (*vide infra*). Pains in the epigastrium and abdomen, which sometimes form the chief annoyance of the patient, usually depend upon passive congestion of the liver (*vide infra*), or upon the tension of the abdominal walls from ascites, œdema, etc.

We must finally mention here the rheumatoid pains in the joints and muscles, from which many patients with heart disease suffer.

The greatest subjective distress occurs in the last stages of heart disease, when general dropsy develops. The patient's helplessness then usually culminates. All motions of the body are difficult, the dyspnœa and oppression in the chest constantly increase, until death finally releases the patient from his mournful condition.

2. **Sequelæ in the Heart Itself.**—We have already discussed the most important sequelæ of valvular disease in the heart itself, its hypertrophies and dilatations. It remains for us to describe the influence of the cardiac disease on the frequency and regularity of the heart's action, and also to discuss some secondary diseases of the cardiac muscle and of the pericardium.

In every well-compensated heart disease the heart's action may for a long time be of approximately normal frequency and regularity. We often find a constant and moderate acceleration of the pulse, which is easily increased from temporary causes. Diminution of the pulse rate (bradycardia) is exceptional in valvular heart disease, except when it is artificially caused by large doses of digitalis. It is most frequent in aortic stenosis. Marked changes in the rate of the pulse are due to severe disturbances of the nervous apparatus of the heart, and hence they are often associated with arrhythmia. Under such circumstances, the pulse rate may be as high as 120 to 140 beats per minute. A rare but interesting symptom is the sudden onset of attacks of enormous rapidity of the pulse, up to 160 to 200 beats and more (tachy-

cardia). This is especially connected with mitral lesions. In the intervals the action of the heart is quiet, with approximately complete compensation of the cardiac lesion. The acceleration of the pulse occurs rather suddenly, and is usually associated with a subjective sensation of palpitation and distress. It may last several hours and then vanish, usually with the same suddenness. The exact cause of these attacks is unknown. They suggest a temporary paralysis of the inhibitory apparatus of the heart. Sometimes a well-marked acute dilatation may be demonstrated by percussion during the attack of tachycardia.

Arrhythmia of the heart is of still greater importance than anomalies of the pulse frequency. It always points to a severe disturbance of the heart muscle (*vide infra*), and perhaps, too, of the nervous apparatus of the heart. In many cases the arrhythmia is the direct result of a simultaneous chronic myocarditis. But in addition to this, the general disturbance of the circulation in consequence of the valvular defect must, of course, make itself evident in the heart itself, and the musculature as well as the nerves and ganglia of the heart cannot remain unaffected by these circulatory disturbances. Hence we generally see marked variations in the frequency and rhythm of the heart's action along with the other signs of beginning disturbance of compensation; but daily clinical experience teaches us that there is not a perfect parallelism between the two symptoms. We find often enough in heart disease a considerable irregularity of the pulse without any of the other signs of marked disturbance of compensation, and, on the other hand, we see in many patients an almost perfect regularity of the pulse up to death. In mitral disease, especially in mitral stenosis, arrhythmia of the heart is much more frequent than in aortic disease.

Recent experimental investigations have resulted in interesting disclosures relative to the causes of irregularities in the action of the heart. Every contraction of the heart begins at the point of entrance of the veins into the auricles, and is then transmitted to the auricles. From the auricles the stimulus is transmitted to the ventricles, probably by means of a bundle of muscle, situated in the auriculo-ventricular septum (the so-called bundle of His). Now, irregularities in cardiac activity are often due to abnormal stimuli originating in the ventricle itself (and, also, but more rarely, in the auricles) that cause contractions which are intercalated between those normally excited from the auricle (so-called extra systoles). After such an extra systole, the next normal ventricular systole is often missing, for the auricular stimulus then strikes the ventricular musculature at a time when, by reason of the preceding contraction, it is unresponsive (the so-called refractory phase). Thus long pauses result (intermittency of the pulse). If we now auscultate the heart and at the same time feel the pulse, we can, as a rule, hear the muscular tone excited by the extra systole, but perceive no pulse beat at the radial, although it can be demonstrated sphygmographically as a slight elevation. We feel, therefore, but one pulse beat to every three or four heart sounds (so-called inefficient heart contractions). But not infrequently, after the first greater pulse wave, a second and smaller one can be distinctly felt (bigeminal pulse, *vide* Fig. 64). Occasionally, and more particularly, as a

Fig. 64.—Pulsus bigeminus

result of the action of digitalis, the entire heart function may assume the bigeminal form for a protracted period; while, on the other hand, there may often be an isolated bigeminal pulse wave at varying intervals in many forms of cardiac irregularity. Besides the occurrence of extra systoles, various other disturbances may interrupt the regular mechanism of the heart action. The most frequent are the so-called transmission disturbances—i. e., disturbances in the normal transmission of the stimulus from the auricles to the ventricles (*vide infra,* chapter on Myocarditis). As a result of these, there is a complete dissociation of the auricles and ventricles, so that the number of auricular contractions (best observed by the number of visible beats of the jugular vein) differs materially from that of the ventricular contractions. Perhaps, too, disturbances in the simultaneousness, strength, and number of the contractions of the two sides of the heart may occur. In short, we see how complicated and difficult is the correct interpretation of all existing irregularities in heart function, and how each individual case can be explained only by thorough study of the heart contractions and the arterial and venous pulse.

Chronic valvular disease of the heart is often combined with anatomical changes in the cardiac muscle, and sometimes in the pericardium.

Among the changes in the cardiac muscle, cloudy swelling, and especially fatty degeneration of the muscular fibers, are the most frequent. The fatty degeneration of the muscle occurs either in a diffuse form, or in little yellowish spots, which are plainly visible on the papillary muscles and trabeculæ. The opinion has often been expressed that fatty degeneration of the muscles is the cause of the disturbance of compensation; that the cardiac muscle performs its increased work until fatty degeneration ensues and reduces its strength. This theory does not entirely correspond to the facts. We have often seen the greatest disturbance of compensation in valvular disease when section of the cardiac muscle showed no fatty degeneration; and, on the other hand, we have seen great fatty degeneration of the heart, as in pernicious anæmia, when there were no signs of cardiac weakness during life. Anatomically, with our present aids to research, we can hardly ever decide with certainty whether the cardiac muscle is still capable of performing its normal functions or not. The usual state of the case is probably this, that fatty degeneration of the cardiac muscle is a result of the disturbance of compensation, and especially of incomplete combustion of the deposited fats because of the defective supply of arterial blood and the diminished muscular activity.

There is as truly a passive congestion of the heart as there is a passive congestion of the liver and kidneys. The circulatory disturbance in the heart itself is the chief cause of cardiac insufficiency, and of the phenomena of disturbed compensation.

A frequent affection of the cardiac muscle in valvular disease is the presence of cicatricial changes and so-called myocarditic nodules in the substance of the heart. Chronic endocarditis may directly invade the subjacent parts of the cardiac muscle and set up a chronic inflammation there, but the cardiac cicatrices usually have another origin. The connective-tissue thickening beneath the endocardium is the result of a simple atrophy of the superficial muscular fibers due to the increased internal pressure of the blood, as in mitral or aortic insufficiency. The connective-tissue nodules within the cardiac muscle, however, depend in part upon a deficiency in the local supply of arterial blood.

Simple sclerotic thickening of the coronary arteries, or complete embolism or thrombosis of a small branch of one of them, is the evident cause of these circumscribed cicatrices. On the other hand, it is also probable that disease of the myocardium is associated with the endocarditis, and is referable to the simultaneous action of the same causes, such as the infection of articular rheumatism, diphtheria, and syphilis. On careful microscopic examination we find, in almost all cases of valvular disease, quite extensive changes in the myocardium (Krehl), and in many cases these may certainly impair the functional powers of the heart. Still, we often find cicatrices of myocarditis without any signs of a previous disturbance in the compensation of the heart. A fuller discussion of this subject will be found in the next chapter.

Pericarditis is not very rare as a result of chronic valvular disease. It is always a dangerous complication, and it may cause death. Regarding its origin, we have found that almost all the cases of heart disease complicated with pericarditis show changes in the aortic valves. Hence it does not seem improbable to us that the secondary pericarditis in such cases is sometimes due to a direct invasion of the pericardium, by the excitants of inflammation passing from the aortic valves through the walls of the blood vessel. On the other hand, the pericarditis may be due to the introduction of excitants of inflammation via the blood current.

3. **Symptoms of Stasis in the Different Organs of the Body.**—As has frequently been mentioned in what precedes, the results of stasis of the blood make themselves manifest in heart disease in various organs. We have already spoken of the important results of blood stasis in the heart itself and the lungs. It remains for us to discuss the symptoms of stasis in the systemic veins.

As soon as the flow of venous blood into the right side of the heart is hindered, the venous stasis is shown by the cyanotic appearance of the patient. This cyanosis may exhibit any degree. In heart disease, which is still, on the whole, well compensated, it is recognized only by the practiced eye of the physician as a slight bluish tinge of the lips, the alæ of the nose, the cheeks, or the nails. With the increase of the disturbance of compensation the cyanosis increases, if it be not modified by the coexistence of general anæmia. In mitral disease, especially in mitral stenosis, the cyanosis is usually more marked than in aortic disease. The large veins, also, become plainly visible as a result of their complete filling, especially the large external jugulars.

A further important symptom which follows the venous stasis is the œdema, the dropsy of heart disease. As we know from general pathology, venous stasis, if it reaches a certain grade, always leads to a transudation of the fluid of the blood from the capillaries. If the lymphatics can no longer carry this transudation away, it collects in the meshes of the tissues and leads to œdema. The œdema of heart disease, therefore, does not appear until the venous stasis has reached a certain degree, and the compensation of the heart disease has become impaired. It first appears in those parts where there is a particularly loose tissue, as in the scrotum, or where mechanical conditions favor its development. The legs usually swell first, especially about the ankles, because here the stasis of the venous blood is increased by gravity. At first, slight œdema appears only temporarily and by day, and disappears again while the patient is in bed at night; but, as the disturbance of com-

pensation increases, the œdema also constantly grows worse, especially in the dependent parts of the body, until finally it may reach the highest degree of dropsy. Besides the œdema of the skin, transudations into the internal cavities occur, especially into the abdomen and the pleural cavity.

The relation between the degree of cutaneous œdema and the amount of dropsical transudation is variable; thus, in particular, we may have a large amount of ascites, with only moderate swelling of the legs. This is probably occasioned in most cases by special secondary changes in the liver (*vide infra*). In other cases the serous exudate, on one or both sides of the pleural cavity (hydrothorax), becomes especially prominent. In short, besides the general circulatory disturbances, local factors (especially the local condition of the vascular walls) appear sometimes to play a rôle. How important a bearing the condition of the blood vessels (aside from the circulatory disturbance) has upon the appearance of œdema is best seen when we compare the occurrence of œdema in different forms of heart disease. How often have I seen juvenile cases of heart trouble with agonizing dyspnœa, cyanosis, hepatic stasis, etc., but with absolutely no œdema of the skin, evidently because the blood vessels were still "tight." On the other hand, œdema appears very early and to a striking degree and extent in older or otherwise predisposed cardiac patients.

The patient's distress is decidedly increased by marked œdema, as has already been said. All the motions of the swollen extremities are considerably impeded. Hydrothorax and ascites increase the dyspnœa, the former by compression of the lungs, the latter by upward pressure on the diaphragm. The passage of urine may be rendered very difficult by œdema of the prepuce. Besides that, we must mention that the skin, when very œdematous, is quite apt to become the seat of furuncular and erysipelatous inflammations.

The results of stasis in the internal organs may be best seen in the liver, spleen, and kidneys.

Passive congestion of the liver is manifested by quite a considerable increase in the size of the organ. The lower boundary of the liver dullness extends several fingers' breadth beyond the edge of the ribs, and the anterior surface and lower border of the liver may often be plainly felt. The liver may also be enlarged in cases in which there are no other marked signs of passive congestion, such as dropsy. Quite severe pain in the hepatic region sometimes arises from the tension of the capsule of the liver. In later stages the liver may grow smaller again through a partial atrophy of the liver cells (atrophic nutmeg liver). Indeed, there may even develop, as a result of the chronic congestion, a secondary cirrhosis of the liver, with a distinctly granulated surface. These are the cases in which marked ascites is especially apt to occur (*vide supra*), sometimes with almost entire absence of œdema of the lower extremities.

Slight jaundice often develops in heart disease, as a result of passive congestion of the liver, or perhaps sometimes from a secondary duodenal catarrh. The peculiar mixture of a cyanotic and slightly jaundiced hue of the skin is very characteristic in many cases, especially in mitral disease. It should be added that the yellowish discoloration of the skin in heart disease is probably not always a genuine icterus, but it may be occasioned by the deposit of other pigments in the skin.

Passive congestion of the spleen arises if the stasis of the blood extends to the splenic vein. The spleen increases in size and becomes firm and dense. It is often hard to make out the congestion from the increase of the splenic dullness, because percussion of the spleen is uncertain if there be also ascites, hydrothorax, etc. We can often, however, plainly feel the enlarged spleen under the edge of the ribs on the left.

In passive congestion of the kidneys, the slowness of the circulation and the diminution of the arterial tension in these organs lead to marked changes in the urine. In particular, there is a diminution in the quantity, especially if the patient is at the same time dropsical. The amount of urine falls as low as 800 to 500 c.c. (27 to 17 ounces), or even less, in twenty-four hours. It becomes dark, concentrated, of high specific gravity, and abnormally acid, and hence it usually has an abundant sediment of urate of sodium. In marked degrees of stasis there is albumen in the urine as a result of the damage done to the glomerular epithelium. The amount of albumen is usually slight, but it may equal one third or one fourth of the volume of urine. Under the microscope we find, in the urine of simple passive congestion, only an occasional hyaline cast, and a few red and white blood corpuscles.

If renal stasis continues for a long time, permanent sequelæ develop in the kidneys; these may be termed "cyanotic induration," or, finally, "congested contracted kidney." By reason of the defective blood supply, the renal parenchyma disintegrates and is replaced by an increased formation of connective tissue (compare, e. g., brown induration of the lungs). The urine in such cases constantly contains albumen, and the chronic disturbance of the urinary secretion is not without influence on the entire picture of the disease. Mild uræmic symptoms may appear, and the disturbances of renal function must have a similar influence on the heart's action (blood pressure, hypertrophy of the left ventricle) to that of primary chronic renal disease.

Careful examination of the urine in cases of severe heart disease is of the greatest practical importance, for the character of the urine, as shown by the color, specific gravity, and amount of albumen, is an excellent index of the vigor of the heart and the condition of the circulation. Any change for the worse in the circulation is directly shown in a diminution of the amount of urine, and an increase in its specific gravity, or, it may be, in the appearance of albuminuria. Any improvement in the circulation, whether spontaneous or due to remedies, is shown first and best by an increase in the daily excretion of urine and a corresponding diminution in its specific gravity.

We not infrequently meet with acute and chronic nephritis, particularly arteriosclerotic nephritis, complicating heart disease. It is often no easy matter clinically to form a correct opinion about such cases.

We may in part refer the numerous gastric and digestive disturbances, such as loss of appetite, vomiting, constipation, and diarrhœa, from which such patients often suffer, to the stasis in the blood vessels of the stomach and intestines; but of course there are not infrequently diseases, such as acute and chronic catarrh of these organs, which are to be regarded as complications.

4. **Embolic Processes.**—The slowing of the circulation, and the disturbances in the nutrition of the walls of the vessels, which result from it, often give rise in heart disease to the formation of thrombi. These are situated in the heart itself, on the diseased valves, in the recesses between the trabeculæ,

in the auricles, etc., or else they form in the veins, especially in those of the lower extremity. From these thrombi fibrinous plugs may easily be set loose and enter the circulation and thus give rise to embolic processes in distant organs. Some of the embolisms, whose clinical relations are especially important, have been more fully described elsewhere, and will be mentioned only briefly here.

Embolism of the pulmonary arteries, proceeding from venous thrombi or from thrombi in the right side of the heart, gives rise to hemorrhagic infarction of the lungs. Its pathogenesis and symptoms have already been discussed in a previous section (see page 333).

Embolism of the cerebral arteries is a common cause of apoplectic attacks, which are not infrequent in heart disease, and usually lead to hemiplegia. The anatomical cause of the hemiplegia in these cases is the embolic softening of the brain which ensues. The details of this are given in the section on cerebral diseases (Vol. II).

Embolism of the larger arteries of the extremities, such as the femoral and the brachial, is much rarer than the forms mentioned. It leads to embolic gangrene of the extremities, unless an adequate collateral circulation can be established. The skin of the peripheral parts, the fingers or toes, first becomes cool, bluish, and at last, if the circulation be wholly checked, almost black. The gangrene advances slowly, usually occupying weeks. Ulcerations develop as the necrotic portions are thrown off. The affection is extremely painful. The patient soon becomes very miserable from the pain and the septic fever that usually attend the ulcerations, and extensive gangrene almost always ends fatally. Sometimes there is embolism of the abdominal aorta. This is usually located at its bifurcation, and occasions a sudden and almost complete paraplegia, speedy disturbance of sensation, abolition of the reflexes, and loss of electrical excitability. No pulse can be felt in the peripheral arteries; the feet are pale and cold, and soon there are signs of gangrene in both legs. The condition is probably invariably fatal.

Embolism of the renal arteries, with consequent anæmic or hemorrhagic infarctions in the kidneys, may have no clinical symptoms at all, but it is sometimes indicated by sudden pains in the region of the kidneys and marked hematuria (see Vol. II).

Embolic infarction of the spleen is often marked by swelling of the spleen and by severe perisplenitic pain. In other cases it is wholly without symptoms.

Embolism of a mesenteric artery is a very rare event. Its symptoms consist of a sudden intestinal hemorrhage, of severe colicky pains, general collapse, and peritonitis.

5. **Complications on the Part of the Nervous System.**—The most important complication on the part of the nervous system—embolic softening of the brain—has already been mentioned. We must also state that cerebral hemorrhage may occur in heart disease. It is especially frequent in aortic insufficiency, either as a result of coexisting atheroma of the cerebral arteries, or perhaps of the abnormally high tension of the vessels during systole.

Mental disorders have been repeatedly observed in chronic valvular disease. They are the result of the disturbance of the circulation and the consequent impairment of nutrition in the brain. Hence they usually make their first appearance in the last stages of heart disease, at the same time with the other

disturbances of compensation. The psychoses in heart disease most frequently have the character of melancholia, but conditions of confusional insanity and excitement also occur.

6. **The Joints.**—Secondary affections of the joints are not rare in heart disease. As acute endocarditis develops in the course of acute articular rheumatism, so, on the other hand, rheumatic pains in the muscles and joints, and even acute swelling of the joints, associated with fever, appear in the course of chronic heart disease. Sometimes we have the complete picture of acute articular rheumatism. Probably no fresh infection from without is requisite for the development of these secondary lesions in the joints, but under certain circumstances there is an unusual development of infectious germs (staphylococci?) on the diseased valves, and large numbers of them passing into the circulation occasion a fresh constitutional infection.

7. **Constitutional Symptoms. Fever.**—In congenital and early acquired valvular disease, the general development of the child is ordinarily much retarded. In heart disease in adults, however, we by no means always see an injurious influence on the general nutrition. In many patients we even find a remarkably good development of fat. A marked general disturbance of nutrition, such as great anæmia and general emaciation, often develops in the later stages, especially in aortic insufficiency. The wasting is, of course, often hidden by the œdema.

In general, chronic heart disease runs its course without fever, but periods often occur in the course of the disease when there is a moderate and usually irregular fever. Marked disturbances of the general condition may or may not be associated with it. The basis of the fever is probably an acute exacerbation of the endocarditis, except, of course, in accidental complications. All gradations occur, from a mild febrile movement without further symptoms to a severe acute recurring endocarditis (*q. v.*). In other cases the fever is connected with secondary swelling of the joints, or with embolic processes.

GENERAL COURSE AND PROGNOSIS OF VALVULAR DISEASE OF THE HEART

The course of valvular disease of the heart is in most cases very chronic. It may last for years. So long as there is a complete compensation, the patient feels almost perfectly well; sometimes he even has no misgivings as to his trouble. The slight difficulty in respiration and the incapacity for physical exertion are noticed, but little attention is paid to them, because the patient is used to them. In other cases there is a moderate disturbance for a long time, but it may be borne quite easily if the patient is rational and prudent in his conduct.

We cannot make any general statement as to the length of the stage of compensation, because cases differ very greatly in this respect. It depends in part upon the intensity of the heart disease, in part upon the external conditions under which the patient lives, and in part, certainly, upon the different individual capacity for work and power of resistance of the heart. Thus it happens that many cases last for decades, while in others severe sequelæ appear within a few months. External injurious agencies, acting on the patient, are of great influence on the course of heart disease. Severe physical

exertion, an injudicious manner of living, intercurrent febrile disease, mental disturbances, care, and anxiety are often followed by unhappy consequences. For readily understood reasons, the difficulties of women afflicted with heart affections are augmented during pregnancy, and similarly, also, the events of labor and the puerperal state not seldom exert an unfavorable influence upon the compensation of an existing heart disease.

If the first signs of disturbed compensation appear, if severe dyspnœa, slight œdema of the ankles, etc., develop for the first time, these symptoms may disappear again completely under proper treatment. Severe disturbances of compensation even, great general dropsy, associated with very weak and irregular action of the heart, may abate, after a few weeks' duration, and the patient may feel quite well again. Exacerbations of the disease may come on several times and as often improve. Finally, of course, the improvement is incomplete. Persistent œdema and the other results of venous stasis ensue, the symptoms constantly increase in severity, especially the dyspnœa, until the patient dies after a long and distressing illness. Immediately before death in heart disease, certain irregularities in the innervation of the heart and in the respiration sometimes develop. Among these the so-called Cheyne-Stokes phenomenon deserves especial mention. It consists in a peculiar periodical variation in the respiratory movements. There will be, first, a complete pause in the respiration (apnœa), and this will be succeeded by feeble breathing gradually becoming stronger, then gradually abating, and finally ending in another complete cessation of the respiration. The patient usually becomes more comatose during the cessation of respiration; his pupils contract. During the hard breathing the patient recovers somewhat, and his pupils dilate again. The chief cause of this rhythmical breathing is probably to be sought in the decided lowering of the excitability of the respiratory center. During the apnœa a considerable amount of carbonic acid collects in the blood before the respiratory center is sufficiently stimulated to resume its activity. We have seen the heart behave in a precisely analogous manner, but independently of any Cheyne-Stokes respiration. There was a series of rapid but regular pulsations, alternating with a series of slower contractions.

In regard to the particular forms of valvular disease, aortic insufficiency generally gives the best prognosis, inasmuch as it may be very perfectly compensated for many years. I know an officer who, with his aortic insufficiency, passed through an entire campaign without any particular difficulty. But if severe disturbance of compensation once occurs in this form of heart disease, it gives a very unfavorable prognosis, since, as a rule, we cannot reinvigorate the heart. Mitral insufficiency is also quite a favorable form of heart disease, which may be compensated for a long time. Mitral stenosis is decidedly more unfavorable in its prognosis, and is associated with more disturbance, but it offers the one advantage that it is relatively most readily influenced therapeutically, especially by proper digitalis treatment. Thus, it may happen that a patient with mitral stenosis repeatedly (half a dozen times and more) experiences the most severe disturbance of compensation, but always recuperates and then for a considerable time is tolerably well. Aortic stenosis is also capable of quite good compensation, but it often causes persistent cerebral symptoms, such as headache and vertigo, depending on anæmia of the brain, or very likely upon simultaneous changes in its blood vessels.

Whether established valvular disease of the heart is curable is a question which cannot be answered unconditionally in the negative. Of course in the great majority of cases it is in itself incurable; only its sequelæ can be prevented or removed to a certain degree. In children and young people, however, cases sometimes do occur, as we ourselves have seen, in which there are all the signs of a pronounced heart disease, but after a long time recovery is complete. Of course it is very hard to decide whether we really have to do with a valvular disease that has been cured, because simple dilatation of the heart, relative insufficiency of the valves, anæmic cardiac murmurs, etc., may easily be confounded with pure valvular disease of the heart.

Among the dangerous intercurrent accidents in valvular diseases we must make especial mention of embolic processes, which may occur suddenly and without warning. The different forms of embolism have been mentioned above, and also the possibility of cerebral hemorrhage in heart disease. The peculiar, and at times life-menacing attacks of cardiac weakness, cardiac asthma, etc., which may occur in all diseases of the heart, will later be more thoroughly discussed.

TREATMENT OF VALVULAR HEART DISEASE

1. **Prophylaxis.**—Our means for preventing the development of heart disease are very limited. To avert endocarditis in articular rheumatism by the present method of treating acute rheumatism with salicylates is impossible. The probability of the onset of endocarditis may be lessened by this treatment only so far as the whole duration of the disease is often considerably shortened.

We can also do little in the way of prophylaxis against the development of heart disease that is chronic from the start, since the cause of the disease is in many cases wholly unknown to us. Those injurious influences deserve the most attention which may favor the development of arterial atheroma and its consequent chronic valvular disease. The chief factors in this connection are excessive physical exertion and an improper mode of living, including the use of too much alcohol and tobacco; but the rôle which these play in the development of genuine valvular disease is certainly much less important than their influence upon certain myopathic and nervous diseases of the heart (see the following chapter).

2. **Treatment of Compensated Heart Disease.**—If we have to treat a heart disease which already exists, but which is at the same time fully compensated, our treatment must be chiefly hygienic. The patient must be made aware of his heart disease without making him needlessly anxious. He must be told that his further good health depends in great part upon his own conduct, his discretion, and his perseverance. The patient must avoid everything which makes great demands upon the heart, or which may have a directly injurious influence on it. All violent bodily exertion, too intense mental work, and also all excesses in eating, drinking, smoking, etc., must be avoided. The use of all alcoholic beverages must be greatly restricted; coffee and tea in moderate strength and quantity may be permitted, if the patient himself does not find them exciting. That the dietetic directions will often collide with the demands of the patient's occupation, as well as with his favorite amusements and his

habits, should not deter the physician from demanding the fulfillment of his prescriptions, at least so far as possible.

Treatment by drugs is usually unnecessary in compensated heart disease. We do not know a remedy which has a directly favorable action on heart disease. The protracted use of iodid of potassium, Fowler's solution, arsenite of antimony ("granules of antimony"), etc., has been recommended. The efficacy of these remedies is very questionable. We can always try them, if a mild disturbance makes a prescription desirable and other remedies are not especially indicated. Beyond this, the physician is usually contented with an endeavor to improve the appetite and nutrition of the patient by means of iron, quinin, and bitters. If there is a suspicion that the heart disease may be due to syphilis, a trial of iodid of potassium may be made, but a brilliant result from the employment of antisyphilitic measures is hardly to be expected, because the mechanical imperfections of the valves, leading to regurgitation and stenosis, can scarcely be remedied.

The employment of baths in heart disease deserves special consideration. Numerous experiences go to prove that they are not only well borne by patients with heart disease, but that they exercise a peculiarly beneficial and invigorating influence upon the action of the heart. In this regard, the greatest reputation is possessed by the warm saline baths, which are rich in carbonic dioxid, particularly those of Nauheim. Even incipient mild disturbance of compensation frequently will be improved by the use of these or similar baths (e. g., Cudowa, Kissingen, Oeynhausen, Soden, etc.). Patients may also employ at home a course of baths with good results. Short, tepid baths with cooler douches sometimes act favorably on the stronger patients. Or salt baths ("neurogen" baths) may be ordered, and especially the artificial carbonic-acid baths, with which many hydriatic institutes are nowadays equipped, but which may also be readily prepared in the patient's home (artificial CO_2 baths of Sandow, Quaglio, Kopp and Josef, and others). The duration of a CO_2 bath should at first be brief (six to eight minutes), and the temperature about 31° to 32° C. (87.8° to 89.6° F.). Gradually the duration may be somewhat prolonged. In recent years there has been much discussion of the treatment of cardiopathies with electric baths (the alternating current baths, the so-called "four-cell baths," and others). Certain favorable effects are not to be denied, though it has not been scientifically demonstrated that a specific action on the heart can be ascribed to the electric baths. At any rate, the inconsiderate and sensational exploitation of electric baths is out of all proportion to their real usefulness, and we must warn against their reckless employment. Where there is no marked disturbance of compensation, the methodical employment of gymnastic exercises, the so-called Swedish movements, has a certain value in the treatment of heart disease. Regular muscular movements promote the circulation of the blood, and thus lighten the task of the heart. If they are carried out cautiously and with a proper consideration of the individual case, they are not infrequently beneficial. Their value should, of course, not be overestimated. The best measure for the amount of bodily exercise for patients with heart disease seems, to the author, to be the subjective sensation of dyspnœa. Any patient who has no special signs of failing compensation may be allowed to walk slowly and also to climb a little so long as he experiences no distinct dyspnœa. As soon as this happens he must

stand still and rest. The author regards it as a great mistake to encourage patients to persist in their efforts regardless of difficulty in breathing. We should not forget that any disturbance of the circulation which occurs must involve the myocardium itself in its influence. See also the remarks made on the treatment of idiopathic cardiac hypertrophy, which apply to valvular disease as well. That a change of climate may prove beneficial is self-evident. It is particularly appropriate that patients with a tendency to bronchitis or rheumatism should spend the winter south.

3. **Treatment of Ruptured Compensation.**—As soon as the compensatory activity of the heart, in a case of valvular trouble, begins to be impaired, as soon as there is marked dyspnœa, diminution in the excretion of urine, and œdema, we must promptly lighten the burden of the heart by complete bodily rest. Many cases, particularly of mitral disease, are completely restored, even when there are marked symptoms of cardiac embarrassment, by mere rest in bed with simple diet and without any other therapeutic measures; but if the disturbance of compensation is more marked and persistent, the physician must have recourse to digitalis, a remedy which possesses undoubted value when the powers of the heart are impaired. Digitalis has the properties of making the separate beats more powerful, of lowering the pulse rate by lengthening the diastole, and above all of heightening the arterial tension. Digitalis is therefore indicated in every case of heart disease when there is persistent disturbance of compensation, and if the pulse is small, of low tension, and, above all, frequent and irregular; the desired effect of digitalis is to make the pulse slower, more regular, and especially of higher tension. Under the influence of the increase in tension thus produced and of the acceleration of the circulation, the disturbances of compensation often disappear in a surprising fashion; there is more abundant diuresis, the scanty, dark, concentrated urine of passive congestion disappears, the daily amount of urine increases, and the urine therefore becomes clear and of lower specific gravity. The œdema then disappears, the dyspnœa ceases, the head becomes free, the general condition improves, and, in brief, there may again ensue a complete compensation of the heart disease. This change is sometimes accomplished in a comparatively short time, in a few days or weeks.

It is very important to prescribe digitalis in a correct manner in order for it to be efficient. Much experience has shown that it is best to give it at regular intervals of three hours, in doses of 1.5 gr. (gm. 0.1) of the powdered leaves, either in wafers or simply in water; so that the patient receives in the course of a day some four or five powders—that is, 6 or 8 gr. (gm. 0.4 to 0.5) of digitalis. If the remedy is well borne we may continue it in this manner, but usually it is omitted during the night, to be resumed in the same way on the next day. As a rule, the distinct specific influence of digitalis is evident after the employment of ten to fifteen powders in the course of two or three days. This is recognized by the marked diminution in pulse rate, the increasing strength and regularity of the pulse, and the improvement in the general symptoms. Instead of the powder we may employ an infusion of the strength of 1 or 2 parts to 150 of water. Of this a tablespoonful must be given regularly every two or three hours. In some cases, particularly if the patient has already become accustomed to the remedy, we may prescribe still larger doses, up to 30 or 45 gr. (gm. 2 to 3) and even more, daily. On the

other hand, we must at times seek to attain the digitalis effect by continued smaller doses (thrice daily gr. jss. [gm. 0.1] or even less). But in every case digitalis should be given in regularly repeated doses, so as to obtain by their combined action the full effect of the drug. It is entirely useless to prescribe digitalis in small doses at long intervals. The remedy must never be prescribed unless we can accurately watch the pulse and the heart's action, for only thus can we obtain clear indications as to the further employment or the omission of the drug.

Not rarely some unpleasant incidental effects appear simultaneously with the beneficial influence upon the heart's action, or with too long continuance of the remedy. Among these are nausea, vomiting, and specks before the eyes. If the digitalis is omitted these symptoms usually cease, while the beneficial effect upon the heart may last for a long time. With regard to these incidental and disagreeable symptoms, different patients vary greatly. Many bear digitalis very well, others very ill. From a therapeutic point of view it is particularly embarrassing if the nausea, vomiting, and other disagreeable symptoms appear before the digitalis has produced an effect upon the heart; but in such cases we should not be too hasty in abandoning the drug, especially if it is urgently indicated. If the patient cannot bear the infusion we should administer the digitalis in powders, or *vice versa*. Or the dosage should be varied, and the digitalis given in smaller, cautious amounts. With sensitive stomachs, the administration of digitalis in the so-called keratin capsules, that do not dissolve till they reach the small intestine, is to be recommended. If the drug absolutely cannot be given by the mouth, it should be injected into the rectum in the form of an infusion of the strength of 1 or 2 parts to 100 of water. This should be warmed to the temperature of the body and given, after a cleansing enema has been employed, once or twice a day. Finally, instead of the infusion, one of the many other digitalis preparations which will be later discussed, will in many cases be resorted to.

Sometimes, especially if the patient has not been carefully watched, the cumulative effect of digitalis is shown in severe symptoms of poisoning, such as collapse, very frequent pulse, disturbance of vision, and dilated pupils. Then such stimulants must be employed as camphor, ether, wine, and strong black coffee. How often digitalis is to be used in the case of heart disease can be determined by experience alone. Many patients, particularly cases of mitral disease, may undergo treatment with digitalis twenty to thirty times or more, with the best results. Every time that signs of impaired compensation return, we must try digitalis again. It must be confessed that it will often be necessary gradually to increase the dose. As is the case with so many other remedies, the patient becomes habituated to the drug. There is no maximum dose, and we must find out by experience in each case what the satisfactory amount may be. Some patients become at last genuine "digitalis eaters," and are unable to exist without large doses of digitalis. We have ourselves seen a patient take 75 gr. (gm. 5) of the powder daily. In other cases it is advisable to continue with smaller doses (about gr. j [gm. 0.05] thrice daily) for some time after the successful administration of the drug in larger amounts. And in other cases, too, cardiac patients with moderate disturbance may occasionally take small doses continuously for a long time (months) with visible benefit. In very many cases, however, the beneficial effect of digitalis even in

the larger doses fails at last to appear. The remedy is no longer borne and it must be abandoned. This usually marks the last stage of the disease.

Not infrequently we see patients with distinct symptoms of passive congestion, in whom the character of the pulse seems at first to contraindicate the administration of digitalis: the pulse is perhaps frequent, but regular and strong; in other cases it is not rapid at all, but yet somewhat irregular; or it may be even infrequent and regular. Particularly in cases of aortic insufficiency is it often very difficult to determine whether we should give digitalis or not. With all patients of this sort it is, in general, worth while to make a trial of digitalis, since it may often be beneficial, and yet we should exercise especial caution and vigilance with regard to its effects.

[Digitalis is used more commonly in this country in the form of the tincture. The urine affords a good guide as to the safety of the continuance of the drug; as long as the renal secretion is sufficient in quantity, and increasing rather than diminishing, there is no danger of the toxic effects. It is, consequently, a good plan to follow carefully the twenty-four-hour quantity of urine when this can be done.

In mitral cases, with or without secondary tricuspid regurgitation, where the cyanosis and other symptoms show that the right heart is engorged with blood which it cannot propel onward, the relief afforded by venesection, or by a dozen leeches in the hepatic region, may be very great. Until the veins are relieved either in this way or by free purgation, digitalis and stimulants are useless, and a resort to them results merely in a loss of time, and perhaps in the loss of a life which might be saved.]

Within recent times many attempts have been made to prepare the active ingredients in a pure form for the purpose of replacing the digitalis leaves. We would mention first the dialyzed preparations (*dialysatum digitalis,* Golaz, and *digitalysatum,* Buerger) of which about 10 to 15 minims each are given thrice daily, generally well borne. Of the active constituents of digitalis, digitoxin is most frequently used in doses of $\frac{1}{250}$ gr. (gm. 0.00025), in pastille form every two or three hours. This is now oftenest employed in the form of digalen (*digitoxin solub.,* Cloetta), which contains mgm. 0.3 ($\frac{1}{200}$ gr.) digitoxin per cubic centimeter.

Generally 0.5 to 1 c.c. (♏ viij to xv) of the solution is ordered t.i.d. The effect is often an excellent one. Digalen may also be used for intramuscular and intravenous injections, especially in cases of marked compensatory disturbances, where all internal medication is futile. The older digitalis preparations (tr. digitalis and vinegar of digitalis) are superfluous and not to be recommended. In general, I am under the impression that digitalis leaves are still the best and most effective preparation, and should be tried first whenever digitalis is indicated. In those rather frequent cases in which this remedy fails, the other digitalis preparations, above all digalen, may prove very valuable. [A new and valuable remedy is digipuratum.]

When digitalis does not yield the desired results, other cardiac remedies are resorted to, partly alone and partly in conjunction with digitalis. Frequently combinations act better than the single drugs. Tincture of strophanthus deserves first mention; its active principle, strophanthin, obtained from the seed of the plant, has almost the same pharmacological properties as digitalis. The tincture is given thrice daily in doses of 5 to 10 minims.

A. Fränkel has recommended intravenous injections of strophanthin ($\frac{1}{200}$ gr. [gm. 0.0003]), injected into a vein of the arm. Upon repeated occasions the author has convinced himself of the strikingly rapid influence of these injections on cardiac activity, but would urge caution, as also with intravenous digalen injections, in employing them in general practice (danger of heart-block). Besides tincture of strophanthus, reference must be made to caffein in the form of caffein sodio-salicylate and sodio-benzoate, given in powders of 1.5 to 4.5 gr. (gm. 0.1 to 0.3) several times daily. Caffein also acts on the vasomotor centers, and is at the same time a diuretic. In this respect theobromin (theobrominsodio-salicylate or diuretin) is even more active, and, especially in combination with digitalis, is often a very valuable remedy. Other heart remedies which have been recommended at times, such as adonis vernalis, convallaria majalis, spartein sulphate, etc., have not proved reliable, and have, therefore, found little acceptance in practice. The valerian preparations are to be considered only in cases with mild subjective symptoms. Camphor is the best stimulant in attacks of acute cardiac weakness. The combination of digitalis with other drugs, usually diuretics, is very useful, as already mentioned. A combination of this sort is the following: Infusion of digitalis, 30 gr. to ℥v of water (gm. 2 to 150); sodio-salicylate of caffein, 30 gr. (gm. 2); tincture of strophanthus, ʒj (gm. 4); liquor potassi acetatis, ℥ij (gm. 60); syrupi aurantii, ℥j (gm. 30). M. S. A tablespoonful every two hours.

4. **Symptomatic Treatment.**—Some symptoms which often occur in heart disease demand a special description.

Dropsy is a symptom of venous stasis, and disappears if compensation be restored spontaneously or by the use of digitalis. Complete rest in bed and elevation of the swollen parts serve as the chief aid in removing the dropsy. Dropsical patients ought also to change their position in bed frequently, if possible, that there may not be too much œdema collected in the dependent portions of the body. It is a good plan to wrap up the swollen arms and legs with flannel bandages under gentle pressure. Mild passage of the œdematous parts may sometimes be of advantage.

The question whether the intake of liquids in dropsical cases should be limited or not is not easily answered. Experience teaches that these patients often lose their œdema when they drink large amounts of so-called diuretic teas (equisetum, eglantine, species diureticæ [P. G.], and others). On the other hand, a material restriction of liquids may be useful (*confer infra*, the remarks regarding to so-called Karrel milk cure). In short, general rules cannot be laid down, and we have to be guided by individual experience. Of internal remedies, besides digitalis, which should invariably be first employed, and drugs of similar action, the true diuretics are to be considered, such as acetate of potassium, acetate of sodium, diuretin, the various diuretic teas, etc. They are sometimes ordered in combination with digitalis (above all, digitalis with diuretin and digitalis with caffein) and sometimes alone, particularly when digitalis cannot be borne or is not indicated. Calomel sometimes shows good effects in cardiac dropsy. Its diuretic influence has lately been emphasized by Jendrassik and others. It is prescribed in powders of 3 gr. (gm. 0.2) three to five times a day. Often a very marked diuresis will be caused after one or two days, with rapid abatement of the dropsy. The administration of the

remedy is stopped as soon as the diuresis begins. It is also omitted if stomatitis develops.

At times it is advantageous to combine calomel with digitalis (digitalis, 1.5 gr. [gm. 0.1]; calomel, 3 gr. [gm. 0.2]), five such powders daily.

In the last stages of heart disease the patient's condition may be particularly distressing from the severe general œdema. It is then necessary to remove the ascites or hydrothorax by puncture, and also to try to relieve the œdematous condition of the skin mechanically.

Scarification of the skin by long incisions into the subcutaneous cellular tissue is effective but dangerous, because erysipelatous inflammation, etc., is apt to ensue at the line of incision. Far more recommendable are little silver capillary trocars (the so-called Southey's trocars, or, still better, Curschmann's trocars), to which a thin rubber tube is attached. Through these trocars, which are introduced obliquely into the subcutaneous tissues, frequently many quarts of serum flow away, drop by drop, so that shapeless swollen limbs become very thin in one or two days. We must always exercise great cleanliness, however, and the utmost disinfection of the skin, wrapping the parts in sterilized gauze. In patients with heart disease it is not, as a rule, advisable to attack the dropsy by sweating, and, at most, it is to be tried only with great caution.

The dyspnœa of heart disease is usually the most distressing symptom of all. Here, too, our chief task is of course to restore the compensation; but this failing, we must try to relieve the dyspnœa symptomatically. Morphin is most efficient in this respect. In general, morphin is, next to digitalis, the most indispensable remedy in the treatment of severe heart disease. It is usually well borne, and procures great relief, especially if given subcutaneously. If we have to do with the last stage of the disease, we need not spare large doses. Otherwise, of course, caution is necessary. Besides morphin, dionin and heroin are used at times.

Chloral hydrate should be cautiously employed in heart disease. It is often, however, well borne, and gives the patient rest and comfort. We have also prescribed chloralamid in cardiac cases, with good results. Where there is absolute insomnia, a trial may be made of 8 to 15 gr. (gm. 0.5 to 1) of veronal, combined, perhaps, with $\frac{1}{6}$ gr. (gm. 0.01) of morphin.

In practice we must often prescribe external applications to the chest, mustard plasters, hot poultices, and also hot foot baths with mustard, ashes, etc. In severe cases their action is slight. Acetate of lead in large doses sometimes seems to have a favorable influence in severe dyspnœa, especially with threatening pulmonary œdema. We give the powder, up to 1.5 gr. (gm. 0.1), every two or three hours, and it is often a good plan to add 0.5 or 1 gr. (gm. 0.03 to 0.05) of opium. We can also frequently obtain decided relief for the patient, particularly if constipated, by a vigorous drastic purge, with compound infusion of senna or gamboge.

Palpitation, constant or paroxysmal, is treated by applying ice to the cardiac region; the "heart bottles," made of tin, or the Leiter heart coils, act very well. In patients with aortic insufficiency and very excited action of the heart, we may recommend the protracted use of ice. Hot poultices also are sometimes beneficial. The narcotics are the most efficient internal remedies, especially morphin, which, of course, we should reserve for severe cases. If the

palpitation is of a lesser degree, we may try bromid of potassium, valerianates, or the like.

The subcutaneous use of morphin is again by far the most potent remedy in the anginous attacks, associated with pain and a feeling of distress. We may also use cutaneous irritation, mustard plasters, etc., hot compresses or poultices, hot hand and foot baths, and internally strophanthus, nitroglycerin, etc. Compare also the section on the treatment of stenocardial and similar seizures in muscular and arteriosclerotic cardiopathies.

We may prescribe bitter remedies—tinctura amara (P. G.), or compound tincture of cinchona—and muriatic acid, for the loss of appetite, in case this is not improved by regulating the activity of the heart. In addition, we must always endeavor to get a regular evacuation of the bowels.

For the attacks of faintness and vertigo, occurring especially in aortic stenosis, as a result of cerebral anæmia, we may prescribe a horizontal position, and stimulants—wine, ether, and Hoffmann's anodyne.

Especial accidents and complications, like pulmonary œdema, infarctions, or apoplexy, are to be treated according to the usual rules.

CHAPTER III

DISEASES OF THE MYOCARDIUM

Preliminary Remarks.—In valvular disease of the heart a disturbance of the circulation takes place because of the functional impairment of the valves, or the obstruction of the orifices of the heart; but in the diseases which we are now to consider we have to deal with impairment of the structure and functional activity of the cardiac muscle itself, while the valvular apparatus of the heart remains intact. The expression myocardial diseases of the heart, in a strict sense, is too narrow, inasmuch as we probably have diseases of the ganglia and nerves of the heart associated with the muscular diseases.

As, however, modern views ascribe great physiological independence to the cardiac muscle, and as our knowledge regarding the pathological changes in the nervous apparatus of the heart is extremely limited, we may, in classifying and considering these conditions, for the present restrict ourselves to the disturbances of heart function observed during the life of the patient, and to the anatomical changes in the heart muscle found at autopsy. In this connection the changes in the coronary arteries must be well considered, as many muscular cardiopathies depend on primary vascular alterations. Since affections of the coronary arteries are, as a rule, connected with changes in the aorta and the other blood vessels, a sharp division between heart disease and vascular disease is impossible.

1. INTERSTITIAL MYOCARDITIS

(Fibroid Degeneration of the Myocardium. Sclerosis of the Coronary Arteries)

Ætiology and Pathological Anatomy.—The anatomical changes of interstitial myocarditis consist in irregular-shaped, glistening white spots, often

very numerous, interspersed through the cardiac muscle where there has been a partial or complete destruction of the muscle fibers and the substitution for them of a firm, fibrinous, cicatricial connective tissue. These places, which are best found by making horizontal sections of the myocardium, occur particularly in the left ventricle, and especially at its apex and in its anterior wall; but they may also be found everywhere, and especially in the papillary muscles.

Besides the ventricles, the auricles must also be carefully examined, as interstitial changes sometimes locate themselves there by preference. Often we may observe fibroid changes as slightly glistening retracted spots on the endocardial or pericardial surface of the heart. The microscope alone, naturally, can give definite information regarding the extent of the disease, and the condition of the muscular tissue.

For the origin of interstitial myocarditis, two viewpoints are chiefly to be considered. In the smaller number of cases it is the sequel to a genuine acute localized myocarditis, such as occasionally occurs in articular rheumatism and other infectious diseases (above all, in diphtheria, but also in scarlatina, typhoid, influenza, etc.).

Here the history sometimes points to such an origin of the disease. But in the majority of cases of interstitial myocarditis there is no inflammation in the limited sense of the term, but we have the sequelæ of a preceding endarteritis (arteriosclerosis) of the coronary arteries of the heart. In places where this change in the vessel causes marked diminution of its lumen, the corresponding portion of the myocardium is imperfectly supplied with arterial blood, and in consequence the muscular fibers gradually degenerate, lose their nuclei, and change into a friable, cheesy detritus. In place of the destroyed muscular fiber there is a new growth of connective tissue. With ordinary endarteritis these processes are slow and gradual, but under certain circumstances there may be a somewhat rapid occlusion of the branches of the coronary arteries, because of thrombosis or embolism of proximal origin. In such cases we have a genuine cardiac infarction, presenting a spot of anæmic necrosis, or sometimes a rather fresh brownish-yellow hemorrhagic infiltration.

Of course, this limited necrosis and interstitial new growth will not occur if, in spite of the existence of arteriosclerosis, the direct supply of blood is still sufficient, or is eked out by the collateral circulation.

If the formation of connective tissue is extensive, the entire heart wall may become decidedly thinner, so that it yields to the internal pressure of the blood. This sometimes occasions in the left ventricle the bulging of a limited portion of the wall of the heart, so-called cardiac aneurism. A cardiac aneurism of this sort, and also an extensive fresh infarction, may exceptionally result in rupture of the heart with escape of blood into the pericardium, and sudden death. Of still greater importance, because far more frequent, is the formation, in places where the fibroid process reaches to the endocardium, of parietal thrombi inside the heart. These sometimes give rise to embolism in distant organs.

As to further pathologic changes in the heart we find that certain portions of the heart are not infrequently dilated or hypertrophied. The dilatation may, at least, in part, be due to the diminished resistant power of the cardiac walls, but in case of hypertrophy we must always seek for special causes, inas-

much as sclerosis of the coronary arteries as such cannot lead to hypertrophy of a portion of the heart. As a rule, the cause is easily discovered, either in an associated arteriosclerosis, or in those factors, such as dissipation, which may occasion, simultaneously with arteriosclerosis, an idiopathic hypertrophy of the heart. Of course, we must also bear in mind the possible but exceptional presence of such complicating diseases as interstitial nephritis, or pulmonary emphysema. With regard to the right ventricle, the fact has weight that it must hypertrophy as a result of congestion in the pulmonary circuit, when the left ventricle is permanently weakened.

The causes of this by no means rare form of myocarditis which we have just described, and which is due to sclerosis of the coronary arteries, are, in general, of course, the same as those of chronic endarteritis (arteriosclerosis). Often the sclerosis of the coronary arteries is merely a part of a general arteriosclerosis, but we sometimes find comparatively marked changes in the coronaries, although there are no specially extensive atheromatous changes in the other arteries of the body, and again, when there is well-marked arteriosclerosis elsewhere, the coronary arteries may show slight symptoms of disease, if any.

Of the various active ætiological factors, the most important are no doubt chronic alcoholism and general dissipation. Furthermore, excessive smoking is certainly a not rare cause, as I have had occasion to satisfy myself, especially in my present eastern sphere of activity. In other cases long-continued severe physical labor seems to favor the development of arteriosclerosis, and clinical experience shows that an important cause of sclerosis of the coronary arteries, particularly, lies in great mental effort and excitement, which indeed are not infrequently combined with the above-mentioned factors—for example, in the case of extraordinarily active business men, speculators, high officials, physicians, and others. All these considerations go to explain the fact that sclerosis of the coronary arteries is seen far more often in the male than in the female sex. That age plays an important part is universally acknowledged. It is true of interstitial myocarditis, as of arteriosclerosis in general, that, as a rule, persons attacked in the second half of life, from forty years of age on. Finally, we must add that hereditary tendencies to the development of arteriosclerosis in general, and sclerosis of the coronary arteries in particular, cannot be denied.

That form of disease of the coronary arteries deserves special mention which is of syphilitic origin, and therefore somewhat distinct from ordinary arteriosclerosis. It is scarcely possible to doubt the occurrence of a specific syphilitic endarteritis of the coronary arteries, usually in conjunction with a general syphilitic endarteritis (*vide infra*), giving rise to almost exactly the same symptoms as ordinary arteriosclerosis. At any rate, this point should in every case be considered, for therapeutic reasons if for none other (*vide infra*).

Secondary chronic myocarditis as a sequel to chronic endocarditis (valvular disease) is only exceptionally of any importance in itself, though of the greatest significance in the clinical picture. The endocarditic process may extend directly to the neighboring muscular layers of the heart, or there may occur, especially in association with aortic endocarditis, embolic infarctions, the development of which is the same as of the previously described thrombotic infarctions of the heart. Finally, there is the possibility that myocarditis and

endocarditis might appear simultaneously, as in polyarthritis and other infectious diseases.

Clinical History.—We must first mention that sometimes quite extensive cicatricial formation may be found in the cardiac muscle *postmortem,* without the occurrence of any manifest symptoms referable to the heart during life. We see, then, that the heart may, under some circumstances, undergo quite a considerable loss in its contractile substance without injury.

In many other cases, however, the heart's capacity for work suffers so much that the same symptoms arise as in valvular disease. The course of such cases may be very chronic. The symptoms begin quite gradually. The patient first has a slight dyspnœa or palpitation, and a feeling of distress and pressure in the chest, but only from external causes, such as slight physical exertion. Sometimes there is marked general weakness and languor. The patients have an unhealthy sallow look. They become easily tired, feel depressed, and scarcely capable of any great bodily or mental exertion. The symptoms gradually increase, and just the same results of disturbance of the circulation appear as in all the other forms of heart disease. The difficulty in breathing becomes more marked, œdema occurs, signs of stasis in the liver, intestines, and kidneys appear—in short, the well-known type of general circulatory disturbance develops.

Physical examination of the heart shows marked anomalies of the heart's action in most of the severe cases. The pulse is often irregular in regard to its rhythm and the intensity of its single beats, but the arrhythmia may also be wholly absent in spite of the degeneration of the myocardium, as we have often convinced ourselves. The pulse is at first quite strong and full, and often, because of the general arteriosclerosis, increased in tension; later it becomes weaker, of lower tension, and at last sometimes very small. As a rule, it is increased in frequency, but we sometimes notice in chronic myocarditis persistent slowing of the pulse to 60, 50, or even less, in a minute. With this slowness of the pulse there is also frequently irregularity of the heart's action, especially the appearance of occasional double beats (pulsus bigeminus).

If persistent and marked slowing of the pulse exists (30 to 40 beats), a careful observation of the auricular pulsations (usually observable in the venous pulsations in the neck) shows that their number is much greater. Frequently it is just double that of the ventricular pulsations (radial pulse). In isolated and rare cases a very extreme slowing of the cardiac beat occurs (down to 16 to 12 beats per minute). Then occasionally there are attacks of unconsciousness with complete cessation of the pulse and respiration, and sometimes also with epileptiform seizures (so-called Adams-Stokes' disease). Percussion usually shows an increase of the heart's dullness, due to dilatation or hypertrophy of the heart, the increase being either general or chiefly on one side. Auscultation shows the absence of any murmur, and hence the absence of valvular disease. The heart sounds are distinctly audible, and sometimes quite loud and sharp, but in the later stages often low and obscure. The pulmonic second sound is accentuated, when there is stasis of the pulmonary circulation. Repeatedly we have found the second sound for a long time very plainly divided (reduplicated). We must also mention that sometimes in pure myocarditis a systolic murmur is heard at the apex which is due either to a relative insufficiency of the mitral valve, or to its incomplete closure, as

a result of defective muscular action of the left ventricle (perhaps more particularly the papillary muscle)—so-called muscular insufficiency of the valve. On the other hand, slight systolic or diastolic aortic murmurs not seldom occur as a result of arteriosclerotic changes.

We must make particular mention of one symptom of sclerosis of the coronary arteries, which, although it is not pathognomonic, is by far most often seen in association with it, and therefore has an important diagnostic significance. We refer to the attacks of so-called *angina pectoris* (stenocardia, heart pang). These attacks of genuine angina pectoris (compare also the following chapter) consist in the sudden occurrence of pain in the region of the heart, extending into the back, the left shoulder, and the left arm even to the tips of the fingers. In severe cases the pain is extremely violent. It is as if the chest were squeezed in a vise. At the same time there is a decided feeling of anxiety and oppression, as well as complete loss of strength—a sense of impending death. The patient seeks some support, he can scarcely move, scarcely utter a few words in a whisper; the extremities grow cool, the brow moist and cold. The attack may be directly fatal, but this is the exception. At a rule, the symptoms pass away in fifteen or thirty minutes, or sometimes only after one or two hours, and the patient gradually recovers. In many cases of sclerosis of the coronary arteries attacks of this sort occur very often, in varying severity. Not infrequently an attack may be due to some special cause, such as the bodily exertion of a long walk or climb, errors in diet, or mental excitement. In such cases bad news may cause death.

With regard to the true essence of angina pectoris, we are confined to hypothesis. The intense pain indicates irritation of the sensory nerves. I am inclined to compare these pains to the severe pains in the extremities accompanying sclerosis of the arteries of the lower limbs and also to many forms of trigeminus neuralgia, likewise associated with arteriosclerosis. Moreover, angina pectoris is by no means alone dependent upon sclerosis of the coronary arteries, but also, without doubt, quite as frequently upon the aortic sclerosis, which is usually present as well (*vide infra*, chapter on Aneurism of the Aorta). The heart's action, as such, may be disturbed simultaneously with the stenocardial attack; then the pulse is small and irregular during the attack. Occasionally, on the other hand, as we have often convinced ourselves, it shows no marked change.

Besides genuine angina pectoris, attacks of cardiac asthma are not infrequent in chronic myocarditis. This differs from angina pectoris in that the peculiar pain and constriction are not present, while the distress for breath, the paroxysmal dyspnœa, is prominent. In most cases of this sort we probably have really to deal with sudden weakness of the left heart.

The general course of the disease varies considerably in different cases. Much depends upon the patient—e. g., upon his ability to take good care of himself. Sometimes dyspnœa, œdema, and other general symptoms of passive congestion are prominent, and in such cases the patient is alternately better or worse. Other cases are characterized by attacks of angina pectoris. The termination is invariably unfavorable. It may either occur gradually with increase in the circulatory disturbance, or with complete suddenness like a stroke.

This important clinical fact of sudden apoplectiform death ("paralysis of

the heart") in patients with sclerosis of the coronary arteries, demands consideration. It usually happens in elderly persons in comfortable circumstances and good livers, who up to that time have not regarded themselves as really ill; but they have repeatedly had slight attacks of vertigo, of oppression, etc. Suddenly a sort of apoplectic attack comes on, often after some definite cause, after a banquet, or after some physical exertion or mental excitement. Death follows in a few moments, or after a deep coma that lasts for several hours or even days. The diagnosis often remains in doubt in such cases, especially if we have not known the patient previously. The autopsy shows, as the sole pathological lesion, a sclerosis of the coronary arteries, with a more or less extensive cicatricial formation in the heart. Apparently in these cases the moment must suddenly arise when the supply of blood to the heart is insufficient, and thus death is caused. Experiments upon artificial closure of the coronary arteries, by Cohnheim and others, agree perfectly with the clinical facts above mentioned. Artificial narrowing of the coronary arteries may also be well borne for a long time, until suddenly both halves of the heart stand still in a condition of diastole. Finally, it should be mentioned that sudden death in sclerosis of the coronary arteries may also be caused by embolism of the trunk of the coronary artery, or, as in one case seen by the author, by the bursting of a focus of myocarditis with hemorrhage into the pericardial cavity.

Diagnosis.—The diagnosis of chronic myocarditis is by no means always easy and certain. We must first determine that there is a cardiac lesion of some kind. This is usually evident from the secondary symptoms of stasis, the condition of the pulse, the area of cardiac dullness, etc. Then the question arises whether we have to do with a valvular disease or with a myopathic disease of the heart. Here auscultation must chiefly decide. The absence of a heart murmur, in spite of other definite signs of heart disease, speaks against valvular disease but not with complete certainty. All murmurs may be absent in the last stages, especially with a high degree of mitral stenosis, and hence we may easily confuse mitral stenosis with myocarditis, particularly when there is marked arrhythmia of the heart. On the other hand we have already stated that in pure myocarditis, with the valves intact, functional murmurs may be present, which may lead to an erroneous opinion as to valvular disease. Chronic pericardial adhesions may likewise be confused with a myocarditis. If continued study of the case has enabled us to exclude valvular disease or an obliteration of the pericardium, it remains to distinguish between chronic myocarditis or sclerosis of the coronary arteries on the one hand, and the other diseases of the myocardium (*vide infra*) on the other. To make this distinction with absolute certainty is not always possible. All the diseases named present the same clinical picture of cardiac insufficiency, but what the anatomical conditions may be which occasion this cardiac insufficiency we can at present only conjecture with more or less probability, during life. If all the signs of a marked general arteriosclerosis (*q. v.*) are present, we will generally not be far wrong in also assuming a sclerosis of the coronary arteries. Of the special heart symptoms, the most characteristic of coronary sclerosis are persistent bradycardia, particularly if associated with arrhythmia and attacks of true angina pectoris. Persistent rapidity and arrhythmia of the pulse occur in interstitial myocarditis, just as in other myopathic diseases of the heart;

but arrhythmia is, in general, far more common when there is actual myocarditis than when there is simple muscular weakness of the heart. There is self-evident difficulty in the diagnosis of cases with sudden apoplectiform paralysis of the heart, and in distinguishing them from apoplexy, embolism, pancreatic hemorrhage, and similar causes of sudden death.

Prognosis.—The prognosis is evident from what has been already said. Recovery is impossible, but even extensive cicatricial formation in the heart may probably last for years without causing much annoyance. We must always be prepared for disturbances of compensation, and the manifold sudden accidents to which patients with myocarditis are exposed, but we cannot foretell the time of their occurrence.

Treatment.—The treatment of chronic myocarditis must be directed first to the dietetic and hygienic care of the patient. This is of the greatest importance. For obese persons accustomed to high living, a moderate, simple diet must be accurately prescribed. Alcoholic beverages must be greatly limited or wholly forbidden, and not more than two light cigars or a few cigarettes allowed *per diem*. It is often best to omit smoking entirely. Moderate bodily exercise is beneficial for the promotion of the circulation and the more rapid diminution of the obesity, but the patient must be earnestly warned against too violent exertion. The best measure of the bodily exercise to be allowed is the subjective condition of the patient. He should cease his exercise as soon as he notices the slightest sensation of oppression in the chest. Neither must mental effort be excessive. In summer, a quiet life in the country or some mountain region is to be advised, or, under suitable circumstances, the cautious use of the waters of Kissingen, Marienbad, Nauheim, Cudowa, etc. Tepid baths, carbonic-acid baths, or saline baths may be employed even at home with advantage. Of internal remedies iodid of potassium has been specially recommended to be used persistently, in the amount of 8 to 15 gr. (gm. 0.5 to 1.0) or more daily. Iodid of potassium enjoys a reputation for an especial specific action upon arteriosclerosis in general, and has perhaps, also, a beneficial effect upon arteriosclerosis of the coronary arteries. We have ourselves seldom seen very striking results from its use, though we frequently give it a trial, particularly if there exists the slightest suspicion of a previous syphilitic infection. Iodid of sodium is also used instead of iodid of potassium, and, particularly of late, sajodin has been frequently substituted. With disturbance of compensation, and with abnormally frequent, weak, and irregular action of the heart, digitalis and similar remedies are indicated, just as in valvular disease. In cases with an abnormally slow pulse, we may use them, provided great caution is exercised, but we must also be governed by the other prevailing symptoms. In severe attacks of angina pectoris (see the following chapter) the subcutaneous injection of morphin is by far the most efficient, and often an indispensable remedy. Further, I recommend, above all, nitroglycerin, which has been greatly lauded by many of my patients. I usually prescribe:

℞ Nitroglycerini ..... gr. $\frac{5}{12}$ (gm. 0.025);
Tinct. valerianæ æther., } āā..... ʒvj (25.0 c.c.).
Spirit. ætheris. nitrosi, }
Dose: 15 to 20 drops.

The nitroglycerin may also be combined with morphin, for example:

℞ Nitroglycerini ..... gr. $\frac{1}{3}$ (gm. 0.02);
Morph. mur. ..... gr. iij (gm. 0.2);
Spiritus, } āā..... ℥ijss. (10.0 c.c.).
Aquæ dest., }

Dose: 20 drops.

Inhalations of amyl nitrite also act favorably at times. For the milder stenocardial symptoms the nitrite of sodium is occasionally useful.

℞ Natrii nitrosi ..... gr. xv to xxx (gm. 1.0 to 2.0);
Aquam dest., ad..... ℥iv (120.0 c.c.).

Dose: 2 to 3 teaspoonfuls daily.

It may also be used in combination with nitrate of potassium, as in the following:

℞ Sodii nitritis ..... gr. xv (gm. 1.0);
Potassii nitratis ..... ℥vj (gm. 24.0);
Elæosacchari menthæ ..... ℥j gr. xv (gm. 5.0).

Dose: A knife-pointful three times daily. Diuretin has also, not infrequently, a decidedly favorable effect upon the stenocardial attacks, and may be given in combination with digitalis, caffein, etc. In cardiac asthma, stimulants are indicated (strophanthus, camphor), but often narcotics as well. Mustard plasters, the application of cold and of heat, hot foot baths, etc., are also employed.

2. SO-CALLED IDIOPATHIC HYPERTROPHY OF THE HEART

(*Functional Strain of the Heart.* [*Heart Overstrain. Soldier's Heart.*])

Ætiology and General Pathology.—Not infrequently cases during life present all the symptoms of an uncompensated cardiac lesion and yet the autopsy discloses merely hypertrophy of the heart with a greater or less degree of accompanying dilatation. There is no lesion of valves, coronary arteries, or myocardium. The cardiac hypertrophy, which involves the left ventricle chiefly, but often both ventricles, cannot be regarded as secondary in the ordinary sense of the word, for in the heart itself and in the other organs we find nothing which can call forth a secondary hypertrophy of the cardiac muscle—no valvular disease, no chronic nephritis, no general arteriosclerosis, and no pulmonary emphysema. Hence we term these cases "primary idiopathic" cardiac hypertrophy, in the sense that we cannot discover any other primary disease.

Notwithstanding, we must in these cases also seek the cause of the cardiac hypertrophy in some excessive demand upon the cardiac muscle, and as there are no macroscopic anatomical or mechanical changes involving an increase in the cardiac activity, we must assume a functional overstrain of the heart.

As a matter of fact, a careful anamnesis often gives us definite grounds for the assumption that there has been functional damage of the heart muscle.

Among the most frequent causes is habitual embarrassment of the circulation by excess in eating and drinking. There can be no doubt that every time food is taken in large amount there is a temporary increase in the activity of the heart, the frequency of the pulse, and the tension in the arteries. This is in part because the specific gravity of the blood is temporarily increased by the material taken into it, and in part because the products of metabolism in the blood have a direct stimulating action upon the heart or the blood vessels, and finally, because an excessive ingestion of liquids temporarily increases the total amount of blood. In brief, there are numerous persons of the more favored classes who indulge too freely in eating and drinking, and for years go to excess in the pleasures of the table; and in the case of such persons there is, during a great part of their life, although not all the time, that condition which the older physicians termed plethora. All these circumstances which we have mentioned occasion an increase in the demands upon the heart, especially its left ventricle. The heart performs this extra work laid upon it, and consequently becomes gradually hypertrophied. From what has been said, it is easy to understand why this form of cardiac hypertrophy is especially frequent in the obese, and particularly in men from forty to fifty years old, though sometimes much younger. We have seen by far the most frequent examples of it in great beer drinkers, and so in persons whose business tempts them to excessive indulgence in beer, such as landlords, brewers, hop dealers, butchers, and many others. Persons of this sort often drink for years, almost daily, four or five, or even eight or ten, liters (quarts) of beer. Let the reader consider what an amount of liquid, and at the same time of nourishment (for one liter of beer contains about 50 or 60 gm. [2 ounces] of carbohydrates), is in this way alone introduced into the blood. This explains the great frequency of cardiac hypertrophy which Bollinger has shown to exist at Munich; but the "Munich beer-heart" is seen with unfortunate frequency in other towns than Munich. The alcohol is probably not a factor in the development of the hypertrophy of the heart, but we may well suppose that it promotes, or at least hastens, the degenerative changes in the cardiac muscle, and particularly in the cardiac nerves, which at last render the heart's action inadequate and the circulation imperfect. For the development of cardiac hypertrophy in itself is a circumstance which does not impair the health, but rather preserves it; only, experience teaches us that no heart can uninterruptedly perform an amount of work which is physiologically excessive, and escape final exhaustion—the point of time depending upon the individual vigor and other associated factors.

A second cause of idiopathic hypertrophy is persistent muscular strain. Muscular exertion always increases the demands upon the cardiac activity. There is an increase in the frequency of the pulse and in the amount of blood passing through the heart. If excessive bodily exertion is habitual for a considerable period of time, there finally develops hypertrophy of the heart, usually affecting both ventricles, but especially the left. Thus is explained the hypertrophy sometimes seen in blacksmiths, locksmiths, baggage carriers, laborers in vineyards ("Tübingen heart"), soldiers after severe campaigns, athletes, and others. Why this hypertrophy should sometimes occur and sometimes be absent, under apparently similar conditions, must be explained by the difference in the physiological capacity of the individual. Here, too,

the abnormality of the condition does not become evident till the functional ability of the heart begins to fail ("heart overstrain," "irritable heart").

In the third place, we must assume that in some few cases of idiopathic hypertrophy of the heart, in which none of the causes yet named are operative, an abnormal nervous irritation of the heart exists which increases its activity (force and rapidity), and hence, finally leads to its hypertrophy. This is perhaps the explanation of many cases of cardiac hypertrophy in persons who have long been exposed to many sorts of psychical excitement. The cardiac hypertrophy of exophthalmic goiter, and the related cardiac hypertrophy often seen in goiter without other symptoms of Basedow's disease (the so-called goiter heart), may also be put in this category. These cases suggest also that perhaps other abnormal products in the body, whose origin may be unknown, might stimulate or harm the heart, and thus finally lead to myopathic heart affections. Especially in women, particularly during the climacteric period, do muscular disturbances of the heart occur. Absolutely no external causes can be demonstrated for these cases, and therefore the hypothesis of some kind of disturbance of "internal secretion" with abnormal effect upon the heart's action suggests itself. To be sure, nothing definite is known on this subject (*cf. infra* chapter on the Nervous Affections of the Heart).

Not infrequently we may assume that several of the above-mentioned causes of cardiac hypertrophy have been operative at the same time. Thus, idiopathic hypertrophy of the heart is seen particularly often in individuals who have to undergo great mental strain and excitement, and at the same time are heavy drinkers, or in those who drink beer to excess and are also at the same time compelled to labor strenuously (brewery laborers, etc.). It is likewise comprehensible that the same injurious factors (mode of living, alcohol, tobacco, etc.) may often produce simultaneously a variety of effects. Thus we frequently find muscular disease of the heart combined with other related diseases (arteriosclerosis, chronic nephritis, gout, diabetes, affections of the liver, etc.).

There is usually associated with the hypertrophy a dilatation, referable to the persistent overdistention of the ventricles during diastole. The majority of cases of functional cardiac hypertrophy belong, therefore, to the type of "eccentric hypertrophy." The weaker and more yielding the myocardium becomes with the course of time, and the more passive congestion affects the heart itself (particularly the auricles and right ventricle), the more the dilatation increases (secondary congestive dilatation); and with it comes degeneration of the cardiac muscle, or, at any rate, functional weakness of the same, giving rise to symptoms of cardiac insufficiency.

Clinical History.—Idiopathic hypertrophy of the heart may certainly exist for a long time without causing the patient any subjective disturbance. We have already emphasized the fact that it is precisely the cardiac hypertrophy which protects the patient for a time from the onset of marked symptoms. Prodromata may occur for a long time before severe disturbance sets in. Among these are palpitation, a certain sense of discomfort in the cardiac region, and slight dyspnœa. The symptoms begin when the heart can no longer respond to the demands made upon it, and when it begins to fail. Then all the symptoms of cardiac insufficiency arise, in just the same way as

in valvular disease and in chronic myocarditis. Hence we need not go into the details of the disturbances of compensation again. The whole series of symptoms of stasis, as well as the attacks of angina pectoris and cardiac asthma, described in the preceding chapter, also occur in idiopathic hypertrophies and dilatations of the heart.

Another subjective disturbance is the peculiar and often very annoying feeling of "fluttering" of the heart—that is, a momentary feeling of fluttering contraction of the heart. This symptom indicates, probably, abnormal conditions of contraction, and must by no means be confounded with the numerous sensations which hypochondriacal and neurasthenic patients refer to the heart. If the action of the heart is irregular, and especially if there is a tendency to bigeminal pulsation, many patients feel every bigeminal contraction as a jolt or stopping of the heart ("stumbling of the heart").

The general course of the disease differs considerably in individual cases. Sometimes there is moderate difficulty in breathing for a long time, especially on any physical exertion. The patient often complains of great languor, or nervous irritability, and sometimes of attacks of vertigo and faintness, and a tendency to perspiration. The appetite is poor, and there is very apt to be constipation. The condition may become quite suddenly worse after any marked injurious influence, especially after great physical exertion or mental excitement. Physical examination discloses all the signs of hypertrophy of the heart. In the first place, the left ventricle is usually enlarged, corresponding to the direct causes of the condition. This enlargement is often better appreciated by accurate observation of the apex beat than by percussion. Later the right side of the heart also becomes hypertrophied; dullness on percussion then extends over the lower part of the sternum, with epigastric pulsation and undulatory movements in the veins of the neck. The heart sounds are pure, at first sharp, later sometimes muffled and feeble. We lay especial value on the characteristics of the first sound of the heart. A dull, indistinct sound is probably a sign of considerable dilatation, while a normal valvular sound indicates a vigorous systole. In general, there is persistent frequency of the pulse, at least in patients who are led to seek the physician because of beginning symptoms. Infrequency of the pulse probably indicates, in most cases, coexistent sclerosis of the coronary arteries (*vide supra*). The frequent pulse may for a long time remain regular, but it is not always so. So long as the left ventricle does its work well the pulse may be tense, and the second aortic sound distinct; but if the heart becomes insufficient the pulse grows small, feeble, often irregular; the heart sounds grow faint, and not infrequently that sign appears which is known as *bruit de galop* or gallop rhythm, and consists in a peculiar reduplication of the first sound of the heart;[1] for each beat of the pulse we hear three sounds over the heart. At the same time the dyspnœa and oppression in the chest increase, the amount

[1] This sign is not infrequently found in idopathic hypertrophy of the heart, in myocarditis, and particularly in secondary cardiac hypertrophy following chronic nephritis. It has not yet been fully and definitely explained. Probably we have to deal with an audible muscle-sound, originating in the auricle, perhaps with peculiar irregularities in the contraction of the heart. Not infrequently we can infer, from the palpation of the apex beat, that a gallop rhythm exists, for the reduplication of the heart beat is distinctly felt. It is also possible that the two ventricles may not contract simultaneously.

of urine diminishes, and œdema appears in the legs. We now have the complete picture of an uncompensated heart disease. With proper treatment the symptoms may disappear again; but, sooner or later, they return. Death finally ensues from general dropsy or from some complications or intercurrent attacks, among which we may mention embolic processes.

Cases of sudden death (paralysis of the heart) from heart failure occur not very infrequently, as we know from experience, in idiopathic hypertrophy of the heart independently of chronic myocarditis.

Diagnosis.—It is by no means an easy task for the physician to make an absolute diagnosis of idiopathic hypertrophy of the heart. Sometimes, when the subjective symptoms suggest an examination of the heart, it is easy to make out an increase in the cardiac dullness and a displacement of the apex beat; but in other cases the presence of emphysema, or obesity, or even of dropsy, makes the physical examination very difficult. If an enlargement of the heart has been demonstrated, we must then exclude valvular disease, especially mitral stenosis, and also secondary hypertrophy, as a result of chronic interstitial nephritis, or other causes. It may be difficult to rule out nephritis if the patient is suffering from passive congestion when he first comes under observation, for then it is often hard to say whether the albuminuria which may be present is to be referred to actual renal disease, or to passive congestion. Even if we can exclude nephritis, we have still to determine whether there is simple hypertrophy of the heart or an interstitial myocarditis. To settle this question is, as we have already said, not always easy. Ætiology is always important—for example, if there has been an excess in beer or in muscular exertion. Frequent attacks of angina pectoris, arrhythmia, and a lowering in the rate of the pulse, suggest sclerosis of the coronary arteries.

The X-ray examination gives very important information. It permits of an almost certain decision, not only as to the size of the heart, but above all as to the condition of the aorta, whether there is marked aortic sclerosis, aneurismal dilatation, or the like. Not infrequently diseases of the myocardium are confounded with chronic obliterative pericarditis. When we consider this latter condition we shall refer again to the differential diagnosis.

Treatment.—The main points in the treatment of idiopathic hypertrophy of the heart are, in general, exactly the same as for valvular disease and interstitial myocarditis. We may, therefore, refer the reader for these to the preceding chapters. A few remarks may be permitted, however, in regard to certain points of view in the so-called dietetic and physical treatment of patients afflicted with heart disease.

1. *Regulation of the Amount of Liquids Taken.*—As is well known, it was Oertel who, a number of years ago, aroused interest in a "new method" of treatment of insufficiency of the heart muscle. He lays particular stress upon the "desiccation" of the body, and especially of the blood. He believes that he can diminish the amount of blood in the body by withdrawing liquids, and thus lighten the task of the heart and restore the normal circulation. On this view rest the proscription of excessive drinking and the limitation of liquid nourishment, such as soup, etc. But, from numerous physiological experiments, we know, however, that the body maintains its amount of blood with great tenacity at a certain constant ratio, for it is able in many ways (secretion and absorption of liquids) to make speedy compensation for varia-

tions arising through changes in the amount of water ingested. It is *a priori* scarcely probable that the total amount of blood in patients with circulatory derangements is increased; and if actually there is a retention of fluid in the body (as indeed is certainly the case where œdema has developed), yet the liquid does not collect in the vessels, but in the lymph spaces of the interstitial tissue, or possibly in the cells of the parenchyma itself. That the total amount of water in the system may be subjected to great variations cannot be doubted. The assumption, however, that in circulatory disturbances there is an increase in the amount of water contained in the blood has been directly disproved by the blood count, by the determination of the specific gravity of the blood, etc. That there should be any increase in the labor demanded of the heart requires that a large amount of fluid should be added to the blood in a relatively short space of time, so that an actual, although extremely temporary, hydræmic plethora should exist. If this process is very frequently repeated, it will surely result in a permanent hindrance to the circulation. These circumstances are, however, actually found only in certain patients, particularly in great beer drinkers. In them, of course, to forbid the ingestion of fluid means to forbid the drinking of beer, and is therefore of the greatest benefit; and we cannot insist too strongly upon the necessity of this injunction in the case of corpulent beer drinkers who are beginning to have some dyspnœa. It is not, however, in our opinion, permissible to apply this rule about liquids, as is sometimes done, to every patient without individualizing. For those who live temperately and are of spare figure, the amount of fluid ingested needs no special attention from the physician. The conditions become decidedly more complicated in heart patients suffering from dropsy, especially if, at the same time, signs of kidney insufficiency present themselves. Here, to be sure, we often get the impression that a decided reduction in the amount of liquid taken assists considerably in the absorption and excretion of the œdema. The "Karell milk cure," recently again recommended by Lenhartz and others, depends upon this principle. The dropsical patient receives for several days no nourishment and no liquid except 200 c.c. of milk four times daily. Under this diet, at times, we note a striking increase in the excretion of urine, and, associated with this, a rapid reduction in the dropsy and the symptoms dependent upon it. This method of treatment deserves consideration in just those cases of muscular insufficiency of the heart that occur in heavy beer drinkers and alcoholics (hotel keepers, brewery hands, etc.). As we shall see later when we discuss diseases of the kidney, the low salt content of the milk probably plays a rôle. Moreover, the dietetic milk cure can be very well combined with the internal administration of pharmacological remedies (digitalis, etc.).

2. *Strengthening of the Cardiac Muscle and Promotion of Compensatory Hypertrophy by Increased Physical Exertion.*—Oertel and others have attempted to incite the heart to more vigorous contractions by means of bodily exertion, and especially by methodical mountain climbing or methodical gymnastic exercises, in order by these means to promote as much as possible the development of cardiac hypertrophy. This view is probably fully justified and of obvious utility in many cases of simple muscular cardiac weakness (*vide infra*). If, however, we apply it to those cases of circulatory disturbance in which there is valvular disease, or some other actual mechanical

hindrance to the circulation, or in which the heart, originally sound, is already suffering from functional strain, the matter seems to be entirely different. For we must consider that we cannot transfer unreservedly to the cardiac muscle our current views relating to the voluntary muscles of the body with regard to exercise and invigoration. The functional activity of the heart is minutely regulated by means of an especial reflex apparatus, independently of our volition. We know that every increased demand upon the heart's activity is in most cases directly fulfilled by an increased cardiac effort. Under proper conditions the most marked cardiac hypertrophy may develop in a completely bedridden patient. We must therefore consider carefully whether, in cases of this sort, the further increase of the demands upon the heart is judicious; whether it may not, on the contrary, contribute to a premature exhaustion of the myocardium. It certainly seems to us that the prescription of increased bodily exertion, such as mountain climbing, should always be given with great caution, and with consideration of the individual circumstances, if the physician desires to avoid unhappy consequences. It may be admitted that a certain measure of bodily exercise is well borne by many patients with valvular and other similar cardiac lesions, but we hold that the benefit lies less in the resultant "invigoration of the cardiac muscle" than in the promotion of the venous circulation occasioned by the motion of the extremities and the deeper inspirations; or, in the obese, in the increased metabolism of fat occasioned by the increased muscular effort. Nevertheless, the inconsiderate prescription of muscular exertion has worked much harm. At any rate, therefore, all attempts to treat heart patients with regular curative gymnastic exercises or methodical mountain climbing must be made with great care and a constant consideration of the individual circumstances. Whenever there are actual signs that an insufficiency of the heart has already developed, complete bodily rest is, in general, much more advantageous for the patient than muscular exertion. A carefully directed mechano-therapy is of use in compensated heart disease, although it frequently can only be regarded as a suggestive remedy. As soon, however, as the muscular exertion is followed by a sensation of respiratory distress or oppression, it must be absolutely stopped. We need not enter here upon the details of the kind of exercises to be prescribed (passive resistance, etc.). The scientific physician will himself be the best judge whether to employ genuine or merely subjective therapeutic measures. The employment of massage is likewise useful to stimulate the circulation in appropriate cases, in which there are such symptoms as slight œdema and moderate dyspnœa.

Bath Treatment of Heart Disease.—Although the different kinds of baths have been frequently utilized in the treatment of diseases of the myocardium, the definite scientific foundation for all these therapeutic measures (CO_2 baths, electric baths, *vide supra,* page 408) is still not very clear. To be sure, we know that the thermic, chemical, and physical stimuli produced thereby bring about certain changes in the blood pressure and vascular tension, which, however, are usually of short duration. We nevertheless must not ignore our practical experience, which in many ways bears favorable testimony to the bath treatment. In practice, therefore, the balneologic treatment of heart disease deserves extended application, although we must avoid being influenced by the exaggerated claims of interested persons. Balneotherapy

may be applied either at certain watering places (above all in Nauheim, but also in Cudowa, Kissingen, Orb, etc.), at sanatoria for the treatment of heart disease, where the various electric baths are particularly used, or at home. For home treatment are especially to be recommended the artificial carbonic-acid baths, to which may be added sodium chlorid, neurogen, pine-needle extract, etc. The baths must always be used with care, and under the strictest supervision. The temperature of the baths should be between 89° to 91° F. (32° to 33° C.). The bath at first should be of short duration (five to six minutes), and later, if necessary, gradually lengthened. After each bath the patient must rest about one hour. Three to four baths are given during the week.

3. HYPERTROPHY OF THE HEART, ASSOCIATED WITH CONGENITAL SMALLNESS OF THE SYSTEMIC ARTERIES

(*Hypoplasia of the Aorta*)

For a considerable time, physicians have occasionally noticed cases, of the sort to be described, in which weakness of the heart and disturbance of the circulation appear at a comparatively early age. The patient complains of palpitation, dyspnœa, and slight œdema. On examination we usually observe anæmia, combined with more or less cyanosis. The cardiac dullness is extended toward the left, and the apex of the heart is displaced in the same direction. There is no marked dilatation of the right ventricle till the later stages of the disease. The heart sounds are perfectly clear, unless there may be a murmur due to relative insufficiency of the mitral valve, or due to imperfect muscular contraction. The heart's action is usually regular, but considerably accelerated; the pulse small and the arteries contracted, but often of high tension. We observe the ordinary symptoms of increasing circulatory disturbance, and finally death. Upon autopsy we find the heart hypertrophied and usually dilated. The valves are normal, but the entire aorta and, in most cases, probably, the other large arteries, are hypoplastic, though otherwise of normal structure. Such cases are termed congenital hypoplasia of the systemic arteries, and it is believed that the hypertrophy of the heart is due to the increased effort to drive the blood through these small vessels.

In our opinion, this condition of the blood vessels deserves full consideration in forming an opinion about idiopathic hypertrophy of the heart; but, ordinarily, still other factors are potent at the same time, for we sometimes find this hypoplasia at the autopsy of persons who showed no material disturbance of the circulation during life, and in whom the heart is of normal or even less than normal size. Virchow has called special attention to the fact that this sort of hypoplasia of the aorta and its branches is sometimes found in association with chlorosis, or, perhaps more correctly, with constitutional anæmia. In these cases the smallness of the reservoir diminishes the volume of blood contained in it, but it does not have any marked influence upon the heart, which indeed has a less than normal burden rather than an excessive one; so that we believe that in those instances of congenital hypoplasia of the aorta in which there is severe circulatory disturbance there is either coincident smallness and feebleness of the heart itself (*vide infra*), or an association of the hypoplasia with other influences unfavorable to the heart. There have been repeatedly observed cases of cardiac hypertrophy due to muscular strain

(for instance, in soldiers), or due to excessive drinking, in which congenital hypoplasia of the aorta and its branches has been found in association with the hypertrophy of the heart; and in the cases which we have ourselves seen and in which the diagnosis was confirmed by autopsy, there have almost always been some other factors unfavorable to the heart besides this congenital abnormality of the blood vessels. Indeed, one can easily understand that the same injurious influences which a normal circulatory apparatus might endure for a considerable time without much damage, would occasion premature symptoms if the vascular system were abnormally developed.

The recognition of hypoplasia of the systemic arteries during life is probably in every case difficult, and scarcely ever absolutely certain. Important factors would be the existence of anæmia and a tendency to shortness of breath and palpitation from early youth; and also the discovery by palpation that the circumference of the arteries was less than normal. The condition of the heart is to be determined in the ordinary manner.

Prognosis and treatment must likewise be governed by the same principles as in other forms of heart disease.

4. PRIMARY WEAKNESS OF THE MYOCARDIUM

(Congenital Weakness of the Heart; Weakened Heart; Acute Muscular Strain of the Heart; Toxic Weakness of the Heart)

Where there is a myopathic heart disease, and the heart is at the same time hypertrophied, this indicates that for a considerable time the heart has done an unusual amount of work. In such cases, therefore, it cannot be that the heart has been feeble from the start. It must indeed have been able to do more than is normally demanded of it. It is not until a later period, when its power is impaired, that we can say that the heart is relatively or finally absolutely feeble. Another group of cases, however, are those in which the heart is originally feeble—that is, its functional powers are below normal. This weakness is certainly, in many instances, congenital. It may be expressed in the structure of the heart, the organ being unusually small with thin walls, or merely in physiological incapacity, the organ being apparently of normal structure. In either case the heart cannot satisfy even the ordinary demands made upon it. Patients of this sort complain, upon the least exertion, of palpitation, shortness of breath, and a sense of pressure over the heart. The pulse is usually frequent or, at any rate, very easily accelerated; a short but rapid walk or a few gymnastic exercises (knee bending, etc.) may raise the pulse rate to 120 or 140 beats per minute.

In many cases there are never any severer symptoms. The patient remains through life feeble, and "with a weak heart"; but if his mode of life is favorable the heart manages to maintain the circulation. In other cases the signs of heart weakness are noticed only at certain times. Of particular practical importance is that frequent form usually called by myself the cardiopathy of adolescence. It occurs in young people between the ages of fourteen and seventeen, when the more rapid growth of the body makes increased demands upon the activity of the heart, and the heart apparently does not keep pace in its development with that of the rest of the body. The patients suffer from frequent palpitation, and even on slight provocation develop a very rapid and

excited heart action, and easily become dyspnœic. It is usually easy to prove by objective examination that there is no organic disease of the heart. The striking symptoms are the excited and rapid heart action, and not infrequently a remarkable hardness of the arteries, apparently strongly contracted in a state of tonus. These symptoms often completely disappear in time; occasionally a certain degree of heart weakness persists, however. The latter condition is particularly evident if increased demands are made upon the heart, as, for example, in the course of military service, or in mountain climbing, or in unsuitable modes of life. Then more threatening symptoms of cardiac insufficiency appear, either suddenly or gradually, and express themselves objectively in dilatation (or stretching) of the heart. In some cases the heart is not able permanently to satisfy even the ordinary demands made upon it. We observe all the symptoms of circulatory disturbance, and at last upon autopsy find a heart which is dilated, but not much hypertrophied. Cases of this sort are not very frequent, but they certainly do occur; their recognition during life, however, is not easy, for it is difficult to distinguish simple dilatation from insufficiency which is subsequent to hypertrophy. We shall probably be able to determine that there is some muscular heart disease because of the increase in cardiac dullness, the smallness and frequency of the pulse, and the ordinary tokens of disturbed circulation, such as dyspnœa, palpitation, œdema, and the urine of passive congestion. If we consider carefully the history of the case and the presence or absence of special ætiological factors, we may be able while the patient is still alive to make a probable diagnosis of simple dilatation of the heart, as a result of muscular weakness.

Acquired muscular weakness of the heart should be distinguished from the congenital (endogenous) variety. It is occasioned by the action upon an originally normal heart of influences which damage the myocardium or its nervous apparatus. We often see temporary conditions of muscular weakness of the heart in anæmia, or following severe attacks of acute disease. Even in these cases there are such differences as to indicate a difference in the original vigor of the heart. The resulting diseased condition is always dependent upon the ratio between the active injurious influences, and the degree of the individual resistance. This law is also evident in acute muscular strain of the heart. Acute attacks of cardiac weakness are seen in the case of soldiers at manœuvers, or in laborious mountain climbing and other athletic exercises (cycling, rowing, football, etc.), such as have of late been so often undertaken in an ill-considered manner; and these attacks are usually associated with acute dilatation. There is a sudden and enormous increase in the demands made upon the heart; the amount of blood which, in a given period of time, must pass through the muscles is increased; and not every heart is equal to the task. A weak heart yields to the increased tension and dilates. The pulmonary circulation becomes congested, the pressure in the arteries falls, and we have all the clinical phenomena of cardiac insufficiency, including dyspnœa, cardiac asthma, and sometimes angina pectoris. If there is prompt cessation of the effort and medical assistance, the condition may be restored to normal and so remain. This, for example, we lately saw in the case of a young and healthy person who escaped drowning only by desperate efforts. But sometimes there remains behind a permanent weakness of the heart, whether because the single excessive strain caused a permanent damage to the heart, or because the heart

was already of less than normal vigor, and betrayed its weakness for the first time when this excessive demand was made upon it.

Certain forms of chronic intoxication are among the causes which frequently lead to acquired weakness of the myocardium, or its nerves. The most important of these, from a clinical standpoint, is chronic alcoholism, the noxious influence of which upon the heart is universally recognized. Less frequent, but still of practical importance, is chronic nicotine poisoning, or, to speak in a more general and perhaps more correct manner, the influence of excessive smoking. This is especially evident in persons who have smoked many strong imported Havana cigars or large quantities of cigarettes. The symptoms consist of an unpleasant subjective sensation in the region of the heart (fluttering, pressure, or palpitation), of slight dyspnœa, and objectively of a frequent, irregular, or intermittent pulse. Other objective cardiac signs are not present, at least at first; but there may be still other symptoms of chronic nicotine poisoning, such as specks before the eyes, disturbance of vision, and dyspepsia. If smoking is stopped in due time, the symptoms may vanish. Otherwise, there follow severer disturbances of the heart, although in most cases there are other ætiological factors also present, such as alcoholism and mental strain. Finally, any extensive anatomical changes (above all, arteriosclerosis) which may have developed in the meanwhile, must be taken into consideration to explain the symptoms.

With regard to prognosis and treatment, there are few special statements to be made. If there are signs of congenital weakness of the heart we must strengthen the constitution in every way possible, and we must seek to give the patient, on the one hand, the proper amount of protection, and, on the other hand, cautious and moderate exercise of the cardiac muscle, by means of medical gymnastics; and in other respects regimen and hygienic precautions are of first importance, from a prophylactic and therapeutic point of view. Carbonic-acid baths, electric baths, etc., have a favorable effect. The treatment of acute and chronic weakness of the heart by such remedies as cardiac stimulants and digitalis is controlled by the ordinary rules. Finally, we may mention in this connection that obliteration of the pericardial sac by chronic pericarditis, or as a sequel of antecedent acute pericarditis (*vide infra*), sometimes occasions atrophy of the myocardium, with resultant feebleness and dilatation of the heart. Cases of this sort may readily be confused with primary cardiac dilatation.

5. THE SO-CALLED FATTY HEART

Ætiology and Pathological Anatomy.—By the name of "fatty heart" we often mean, at present, two quite distinct conditions of the heart—the one an abnormal deposit of fat in the heart, and the other a fatty degeneration of the muscular fibers of the heart.

Fatty overgrowth and fatty infiltration of the heart are usually merely symptoms of great general obesity. At the autopsy of very fat people we sometimes find the heart entirely inclosed in a thick capsule of fat. The fat is situated chiefly in the external pericardium and beneath the visceral pericardium. It is usually abundant along the course of the larger vessels within the grooves of the heart, but in marked cases the fat also involves the muscular

substance, so that many groups of fat cells are interspersed between the muscular fibers. The heart itself is otherwise quite normal or somewhat hypertrophied or dilated. There are in some instances also present sclerosis of the coronary arteries and indurations due to myocarditis.

We have already mentioned fatty degeneration of the muscular substance of the heart as a frequent result of valvular disease. In myocarditis and idiopathic cardiac hypertrophy, and in the hypertrophy secondary to chronic nephritis and pulmonary emphysema, we also meet with fatty degeneration. We often find fatty degeneration of the heart, as well as of other organs, in severe acute infectious diseases, in phosphorous poisoning, and in all marked primary and secondary anæmias. Under the microscope we find the muscular fibrillæ studded with little drops of fat, which may be so numerous that the nuclei and the transverse striation of the fibers are quite concealed by them. In all severe cases of fatty degeneration the contractile portion of the heart muscle also suffers; but investigations into the details have not yet been reported.

If the fatty degeneration is of high degree, we can easily recognize it with the naked eye. Beneath the endocardium, especially on the trabeculæ and papillary muscles, we see very fine and delicate yellow points and striæ. With great fatty degeneration, as in phosphorus poisoning and pernicious anæmia, the whole cardiac muscle is manifestly yellow, and also soft and flabby. It is claimed that rupture of the heart may occur as a result of marked fatty degeneration.

Hitherto it was usually believed that in fatty degeneration of the heart muscle the fat resulted from the decomposition of the albumen in the muscle cells. According to newer investigations it has become probable that the fatty infiltration of the muscle fibers is only the visible expression of a deficient fat oxidation, following insufficient muscular activity. Upon this assumption, the fat does not originate in the muscle fibers, but it is brought to the myocardium from the fat depot of the body (the subcutaneous cellular tissue).

Clinical Symptoms.—Fatty degeneration of the heart has no characteristic clinical symptoms. In the conditions under which we know it is apt to occur, we can usually suspect it during the lifetime of the patient, but we cannot diagnosticate it. We must also mention that the frequently expressed opinion, that fatty degeneration of the heart invariably occasions general cardiac weakness, is very often incorrect. In pernicious anæmia there may be quite a strong and a perfectly regular pulse up to death in spite of the most marked fatty degeneration, and many personal observations leads us earnestly to dispute the view that fatty degeneration of the heart is to be regarded as the regular cause of cardiac insufficiency, and the consequent disturbances of compensation. We have made microscopic examinations in many cases, and failed to find any sign of fatty degeneration in the myocardium, although during life the signs of cardiac insufficiency were well developed. In general, with our present knowledge, it is impossible to establish definite relations between the histological condition of the muscular tissue of the heart and its functional activity during life.

We cannot say much that is certain in regard to the clinical symptoms of a deposit of fat in the heart. "Fatty degeneration of the heart" always plays a far larger part in popular speech than it does in reality. It is certainly a

fact that difficulty with the heart and respiration is very often observed in fat people. Examination of the heart, which, however, is decidedly impeded by the thick panniculus adiposus, often shows in such cases an increase of the cardiac dullness, a small and sometimes irregular pulse, and faint but clear heart sounds. The disturbance may be very considerable, attacks of angina pectoris and cardiac asthma may come on, and death may follow with increasing dyspnœa and general œdema.

If one has opportunity to make an autopsy in such cases, there will be found no single, constant anatomical change as the cause of the cardiac disturbance, but usually idiopathic hypertrophy (*vide supra*), or, less often, myocarditic changes with sclerosis of the coronary arteries, and the like. Sometimes, but by no means invariably, there is, of course, a marked deposit of fat upon the heart itself, but the question arises whether this can directly and seriously embarrass the cardiac activity. The fact is that we often have seen similar well-marked cases of fatty heart which during life presented no special cardiac symptoms. There would be more reason in ascribing an unfavorable influence to the fatty infiltration of the cardiac muscle; but in such cases there is almost always a coincident atrophy of the muscular structure, so that it is questionable whether the fatty infiltration is to be regarded as actually the primary pathological process. We ourselves are far more inclined to the view that there is primary atrophy of the myocardium, with this fatty infiltration as a secondary phenomenon, similar to the frequent and well-known occurrence of secondary lipomatosis of atrophic voluntary muscles. At any rate, we are as yet entirely unable to recognize cases of this sort of fatty heart during life, and it is certain that they are much less frequent than the other myopathic diseases of the heart.

Therefore we cannot associate with the term "fatty heart" any uniform anatomical and clinical conception. It would be better to speak of the "heart of obesity"—that is, of all the manifold injuries to which the heart of obese persons is exposed.

For the same conditions that lead to general obesity (luxurious mode of life, excessive beer drinking), produce, in themselves, usually an injurious effect upon the heart. Just what particular form of heart disease is present in a case of obesity, must be carefully considered in each individual case. Without doubt, the most frequent form is the simple "idiopathic" cardiac hypertrophy (the so-called "beer heart," *vide supra*). The arteriosclerotic heart disease is rarer. At any rate, physicians should have more regard for the facts of pathology, when they are inclined to make an off-hand diagnosis of fatty heart.

Treatment.—A great part of the disturbance of respiration in fat people depends not upon the cardiac weakness, but on the corpulency itself. The great bulk of the body, and the hindrance to the activity of the respiratory muscles, are very important factors. Treatment directed against the respiratory disturbance must hence attack the obesity chiefly, and thus in many cases we shall also assist the action of the heart. The detailed description of the hygienic methods of cure to be employed here is to be found in the chapter on Obesity (*q. v.*).

In regard to the special treatment of the cardiac symptoms in the obese, this does not differ from the rules that obtain in other forms of heart disease.

CHAPTER IV

THE CARDIAC NEUROSES

Our medical experience teaches us that many people complain of really serious cardiac symptoms who, on the most careful objective examination, show no trace of organic disease of the heart (valvular disease, dilatation, hypertrophy). Occasionally only purely subjective sensations are present, such as the feeling of pain, pressure, or distress in the cardiac region. Frequently, however, functional changes in the cardiac action are at the same time present, as, for example, permanent or temporary frequency of the pulse, irregularities of the heart beat, etc. At the present time we are accustomed to group all these cases under the general heading of "cardiac neuroses." In their development, however, quite a number of factors seem to require consideration. So far as my personal experiences are concerned, I would like particularly to emphasize the following aspects:

1. **Purely Psychogenetic Heart Disturbances** (i. e., subjective and objective disturbances resulting from a primary groundless fear, of any origin whatever).—It may be said without reserve that the number of cases of "imaginary heart disease" is decidedly greater than the number of cases of actual organic disease of the heart. The primary factor is the fear of a heart lesion and its consequences. The numerous discussions on disease, the reading of medical pamphlets and announcements, often also the tales of members of the family or acquaintances who have suffered from really serious heart disease, arouse a lively fear of becoming victims to the malady in many timid persons. This primary change of consciousness produces, first, a variety of autosuggested subjective sensations in the cardiac region, and secondly, marked disturbances of the cardiac activity, such as tachycardia, isolated extrasystoles with intermittent pulse, etc. As these primary groundless fears are not continuously present in the consciousness, the cardiac disturbances not infrequently appear in attacks. In many cases they occur with preference at night, partly because the mind is no longer diverted by the business of the day, and partly, in all probability, because they are sometimes induced by harrowing dreams. The patients then awake suddenly with marked sensations of fear, palpitation, and distress. The diagnosis of the psychogenetic disturbances of the heart is generally not difficult. The nature of the complaints and the general neurasthenic condition of the patient soon show the experienced physician the actual state of affairs. Notwithstanding this, a careful objective examination is, of course, always necessary. The treatment must be primarily a purely psychic one. The quieting of the patient, the sympathetic reassurance that no serious malady is present, is the main thing. In practice, to be sure, the suggestive influence of other remedies (valerian preparations, CO_2 baths, hydrotherapy) cannot be dispensed with Actual cardiac remedies, such as digitalis, should be avoided. As the nervous heart symptoms are often only partial phenomena of a general neurasthenia, the reader is referred also to the chapter on this subject in Vol. II.

2. **Nervous Heart Disturbances from Endogenous Toxic Influences.**—In all probability the influence upon the heart and its nerves of certain endogenous

toxins which develop in the process of tissue metabolism is of great importance, although this subject has as yet been by no means exhaustively investigated. The influence of the thyroid gland is best and longest known. We know that in exophthalmic goiter very decided changes in the heart's action can be produced by the abnormal thyroid activity. Besides those met with in fully developed cases of Basedow's disease, there are also numerous other disturbances of the heart that are probably dependent upon morbid changes in the thyroid (so-called goiter heart). At any rate, this possibility must constantly be considered and the treatment arranged accordingly, should an enlargement of the thyroid be present (thyroidin tablets, Möbius's serum iodin, X-ray treatment). To be sure, with our lack of knowledge of the actual processes, the proper determination of the indications is very difficult, and we are therefore forced to careful experimentation. Further details concerning these important matters will be found in the chapter on exophthalmic goiter. It appears to me very worthy of consideration that perhaps other organs besides the thyroid gland may, when diseased, disturb the heart's action. Primarily, in my opinion, we must consider the female genital organs. The "nervous" heart symptoms so frequently developing in the menopause are perhaps, in part at least, to be interpreted in this sense, as are also the heart disturbances in cases of uterine myomata, and those following bilateral oöphorectomy. There is a particular variety of muscular heart disease with arrhythmia, that I have observed only in women. I am therefore inclined to associate these cases with this type of disturbances of the internal secretion. Occasionally in men, nervous heart disturbances appear in connection with diseases of the prostate. It must be mentioned that in all the cases apparently belonging to this group, general nervous and psychic disturbances appear. The cases of exophthalmic goiter are the best demonstration of this fact. Finally, the influence of abnormal digestive processes on the heart's activity is worthy of notice. In certain cases abnormal stimulation emanating from the stomach or intestines seems to affect the heart. These cases are not particularly frequent, and their interpretation is by no means always perfectly clear. Toxic as well as reflex influences may play a rôle. Sometimes, perhaps, it is merely the mechanical pressure upon the heart from the dilated stomach or colon.

3. **Nervous Heart Disturbances Due to Exogenous Toxic Influences.**—In this class are to be included those cases where definite external poisons are to be regarded as the cause of the existing heart disturbances. Most important in this group is the tobacco-heart neuroses of heavy smokers; those induced by lead, alcohol, etc., are less frequent. We must further include the nervous heart affections subsequent to acute infectious diseases (influenza, diphtheria, typhoid, etc.). We must, however, consider that just in these cases the drawing of the line of demarcation between nervous disturbances and organic heart diseases, such as myocarditis and arteriosclerosis, is often very difficult.

From what has just been said, it is obvious how difficult a really exact diagnosis of nervous disease of the heart often is. Each case must be judged from the individual circumstances. In the suggestions given above, the most important diagnostic factors, at least, are indicated.

4. **The Nervous "Attacks" of Patients with Heart Disease.**—Under certain conditions, in almost all the forms of heart disease, particular symptoms

may develop paroxysmally. These attacks form but a part of the general clinical picture.

Occasionally, however, such attacks may also develop apparently independently with otherwise normal cardiac signs. They then form a particular variety of "nervous heart disease" or "heart neurosis." On account of their practical importance, we take the liberty of inserting a few special remarks relative to these attacks.

(*a*) *Attacks of Simple Heart Weakness Associated with Feebleness and Smallness of the Pulse.*—The latter is usually rapid, and frequently irregular and diminished in tension. Along with these symptoms are sensations of distress and dyspnœa (cardiac asthma).

(*b*) *Attacks of Stenocardia, or Angina Pectoris.*—The characteristic feature of attacks of stenocardia is an oppressive, tightening sensation, that is felt occasionally in the heart region, but usually in the front of the chest behind the sternum. The pain may become very severe. It often radiates to the left shoulder and arm, down to the elbow, and even to the finger tips. The pain may also radiate to the right arm, or simultaneously to both arms, and sometimes upward to both sides of the neck. Associated with this pain is an unusually marked feeling of weakness, as if all strength were lost ("feeling of impending dissolution"). Sometimes the arms feel as if dead. Actual dyspnœa is not always present, but there is often a sensation of fear and distress. The face is usually pale, not really cyanotic. During the attack, the pulse is sometimes only slightly changed; in other cases, its frequency is increased, less often diminished, or it is irregular. The duration of such an attack is often only a few minutes, but sometimes it may last half an hour or more. Not infrequently, at the end of the attack still other symptoms appear, such as belching, vomiting, perspiration, and the excretion of a profuse, light-colored and dilute urine ("spastic urination").

Concerning the actual processes in an attack of stenocardia, we know little that is really definite. I believe that the symptoms are best explained by the assumption of a vascular cramp, and that the pains actually occur in the vessels themselves. The sternal pain is an aortic pain, and the radiations of the pains into the arms and neck, etc., depend upon the involvement of the brachial, carotid, and other arteries in the spasm. The variations in the symptomatology of the anginal attacks are explained by the different localizations of the cramps.

[For the very plausible "viscero-sensory reflex" theory of anginose pain, see James Mackenzie's "Diseases of the Heart."]

True angina pectoris occurs principally in sclerosis of the aorta (*q. v.*), and in sclerosis of its branches, the coronary arteries, etc. I have seen attacks with pallor and coldness of the arm which could only be explained by a spasm of the markedly sclerosed artery of that extremity. The comparison between angina pectoris and the attacks of intermittent claudication (*dysbasia arteriosclerotica*) is equally obvious. In certain cases we must also think of a participation of the abdominal aorta and its branches, giving rise to abdominal pain, sudden tympanites, and disturbances in the function of the intestine, kidney, etc. It is possible that true angina pectoris may also occur in other organic diseases of the heart, without simultaneous arteriosclerosis, but this, to my knowledge, has really not been definitely proved. Nevertheless, some

authors (Nothnagel) have assumed the existence of a purely nervous or "vasomotor form of angina pectoris." It is not impossible that the anginal attacks of heavy smokers may be of nervous origin, but just in these cases the existence of an aortic sclerosis can scarcely ever be excluded with absolute certainty. In true angina pectoris the most important exciting causative factors are bodily exertion, further, marked mental excitement, and errors in diet.

In general, the diagnosis of angina pectoris is not difficult if one bears in mind the symptomatology described above. It cannot be denied, however, that frequently attacks occur in heart disease that cannot be definitely classified. The boundary between stenocardia and cardiac asthma is often very obscure. We must also guard against confusing organic disease of the heart with hysterical attacks. The prognosis of true angina pectoris is always grave, although some patients suffer from their attacks for many years. The danger of a sudden fatal termination is always present.

In the treatment of severe attacks of stenocardia the most useful remedy is a subcutaneous injection of morphin. It often gives prompt relief from the most agonizing symptoms. The internal administration of morphin, although less rapid in its effect, is also useful. Next to morphin, I consider nitroglycerin the best remedy. It is best given in drop form, as in the following mixture: Nitroglycerin, gr. $\frac{1}{3}$ (gm. 0.02), and spirits of wine and distilled water, of each ℥ijss. (gm. 10.0). Of this, 20 drops are to be given to the dose. To this solution 3 gr. (gm. 0.2) of morphin hydrochlorid may be added. Many patients obtain relief from the inhalation of amyl nitrite. Diuretin is another useful remedy, but as it does not act promptly, it is more to be used as a prophylactic against the attacks. Aside from the remedies mentioned, mustard applications to the chest, hot hand and foot baths, and hot applications to the chest, and the like, have also proved serviceable.

(*c*) *Attacks of Fright, Anxiety, and Distress on Going to Sleep.*—A very characteristic symptom in some serious cases of heart disease is the sudden occurrence of a severe feeling of distress when the patient is about to fall asleep. As a rule, this depends upon a kind of Cheyne-Stokes symptom (*vide supra,* page 406—with a cessation of respiration, so that the patient awakes suddenly in a great fright.

(*d*) *Attacks of So-called Humid Asthma.*—These are attacks associated with a very rapidly developing profuse serous or serosanguineous exudation into the bronchi and a consequent marked dyspnœa with profuse frothy serous, and usually somewhat bloody expectoration. Sometimes the exudation into the bronchi is associated with a similar process in the gastro-intestinal tract, which shows itself in vomiting and profuse diarrhea. All these symptoms suggest a temporary vascular paralysis with osmosis of serum through the walls of the vessels, which have become porous. The local dilatation produces anæmia of the brain, and therefore, at times, the mind becomes clouded, or even complete loss of consciousness occurs. During the attack the pulse becomes very weak and small. These attacks occur in arteriosclerotic and myocarditic heart disease, and, above all, in the heart affections associated with nephritis. The most important indication for treatment is the energetic use of cardiac and vascular stimulants, such as subcutaneous

injections of camphor, digalen, and caffeine; and also sinapisms, hot applications, etc.

(*e*) *Attacks of Tachycardia.*—(*Vide infra.*)

5. **Nervous Palpitation.**—By "palpitation" we understand the subjective perception of the movements of the heart. It is usually excited by increased action of the heart, but there is no constant relation between the intensity of the cardiac pulsations and the subjective feeling of them. We sometimes observe that patients with aortic insufficiency do not perceive the very strong action of their hypertrophied hearts, while in other cases a patient complains of a troublesome feeling of palpitation, although the action of the heart does not appear objectively to be especially increased.

We term cases "nervous palpitation" if the patient complains of palpitation when a physical examination of the heart shows no anatomical change in it. As a rule, in these cases we really have to do with a heart whose action is increased by abnormal nervous influences. In many cases the palpitation arises from slight external causes, which may give rise to little or no palpitation in a healthy person, as, for example, after the slightest mental excitement, after any slight physical exertion, after taking food, after indulging in certain drinks, such as tea, coffee, wine, or beer, or after assuming certain positions, as in lying on the left side. Here, then, we have to do with an abnormal sensitiveness of the heart to external irritation. Usually the increase in vigor of the heart's action is associated with a decided increase in its frequency. We have often seen patients with nervous palpitation who had a pulse rate of 140 to 160 beats a minute after comparatively slight exertion; but in other cases there is apparently a sort of hyperæsthesia of the patient with regard to the motions of the heart, so that contractions of normal strength and rapidity give rise to disagreeable sensations.

The patient rarely complains of continuous palpitation; it usually occurs in more or less sharply defined paroxysms. Very commonly in purely nervous palpitation we have to do with persons who, in general, suffer from nervous, hysterical, and neurasthenic symptoms, or they are anæmic persons, chlorotic girls, etc.; but, on the other hand, nervous palpitation may occur in very full-blooded, "plethoric" subjects.

Often hypochondriasis plays an important part, and dread of heart disease and the thought of its possible consequences excite a palpitation which confirms the patient in his delusion (*vide supra*, Psychogenetic Heart Disturbances).

The diagnosis of nervous palpitation can be made only when repeated careful examination shows no objective abnormality in the heart. In many cases, as when there are anæmic murmurs, the decision may be quite difficult. We must always pay particular attention to the whole constitution and the general impression which the patient makes.

It is often especially difficult to distinguish between purely nervous palpitation in a heart otherwise functionally normal, and conditions of congenital or acquired weakness of the heart (*vide supra*).

The prognosis is so far favorable in that the disease is not dangerous. In many cases improvement and final recovery may be effected, but other cases, of course, resist all therapeutic efforts very obstinately.

The treatment must first be directed to improving the patient's general

constitution. The anæmic are to be given iron, quinin, and strengthening diet. We put full-blooded persons, however, on scanty fare, and prescribe for them bitter waters, or a bath cure at Marienbad or Kissingen. When there is hysteria or neurasthenia, it requires special treatment. If the patient is hypochondriacal, which is the case in the majority of "cardiac neuroses," of course the main thing is for the physician to reassure him. At the same time, we must not disregard entirely the use of other remedies, although they may be of suggestive value only. The actual heart drugs, however, such as digitalis, are best avoided completely. The most useful remedies are hydrotherapy, CO_2 baths, and the internal administration of tincture of valerian, validol, bromid of sodium, etc.

As a symptomatic indication during an attack we should recommend the patient especially to keep quiet. The use of cold to the cardiac region—cold compresses and ice bags—often acts beneficially. On the other hand, however, it is to be noted that a tendency to palpitation associated with cardiac weakness may often be allayed by methodical exercise and the invigoration of the cardiac muscle consequent thereupon.

6. **Paroxysmal Tachycardia.**—A peculiar and not very rare neurosis of the heart, tachycardia, consists of an enormous frequency of the pulse (so-called heart hurry), coming on in paroxysms, up to 160 to 200 beats and more a minute.

We have already mentioned these attacks as a symptom in mitral and aortic valvular disease and in diseases of the myocardium, but precisely similar attacks occur as a pure neurosis without demonstrable lesion of the heart, particularly in anæmic, nervous, or obese individuals. We saw one very well-marked case in a lady with extreme neurasthenic melancholia, and other cases in patients with the greatest variety of other diseases, such as cirrhosis of the liver, multiple sclerosis, etc. We do not know the exact cause of the sudden development of the increased pulse frequency. Sometimes heavy smoking seems to be responsible; in other cases, bodily exertion, mental excitement, and previous acute diseases appear to have an ætiological significance. Not infrequently, absolutely no cause for the appearance of the tachycardia can be demonstrated. The individual attack usually begins quite suddenly, by day or by night, sometimes without any cause, but often it is apparently produced by certain exciting causes, especially at times by overdistention of the stomach. The patient feels that the attack is coming, he becomes anxious and restless, and looks pale; but there are not, as a rule, any symptoms like præcordial anxiety, dyspnœa, or attacks of faintness. The majority of patients lie down quietly during the attack, others walk about slowly. We notice in the heart itself, during the attack, chiefly a great acceleration of the heart sounds. Both heart sounds become completely similar in volume and pitch, and the period of repose of the heart disappears ("embryocardia"). We sometimes hear indefinite, functional murmurs. The action of the heart is usually quite regular, but there is occasionally manifest arrhythmia during the attack. Acute increase of the heart's dullness has been repeatedly observed during the attack. We have been able to verify a marked temporary dilatation of this sort, particularly in patients with heart disease, and also in one case of paroxysmal tachycardia affecting a sufferer from cirrhosis of the liver. In the majority of cases, and especially when the

tachycardia is a mere neurosis, no such dilatation of the heart can be made out. As a rule, the respiration is not disturbed during the attack. Sometimes the excretion of urine is diminished, but occasionally during, and particularly after, the attack, polyuria may develop. Gastric disturbances are not infrequently observed (anorexia, nausea, vomiting). The duration of the attacks is very variable, ranging from a few moments to several days. If frequent opportunity exists to observe the end of the attack, usually a sudden cessation of the tachycardia is noticed in testing the pulse or auscultating the heart. It is noteworthy that the frequency of the pulse usually sinks to exactly one half of the previous count; for example, from 180 suddenly to 90. This is extremely interesting from a theoretical standpoint, as it permits us to regard the attack itself as a duplication of the heart beats.

The prognosis of tachycardia depends first upon the nature of the underlying disease. We do not know whether a permanent recovery is possible in idiopathic cases, but we can, at any rate, succeed in improving the condition. If no other signs of organic disease are present, life is not actually threatened by the attack. The treatment during the attack consists in enjoining complete bodily rest, and in applying ice to the heart. Such sedatives as bromid of potassium, water of bitter almonds, and tincture of valerian are particularly beneficial in the "purely nervous" cases. The application of a cold pack is usually acceptable to the patient. It is said that compression of the vagi in the neck has caused the tachycardia to disappear in isolated cases. In other respects our treatment is determined mainly by the underlying condition, if known. [If there is reason to think that the stomach is overloaded, gentle measures should be taken to empty it.] The main thing in all cases associated with general neurasthenia and nervousness is a suitable constitutional treatment (hydrotherapy, a sojourn in the country or in the mountains). Precise regulation of the diet, according to the patient's constitution and mode of life, is likewise necessary.

Concerning bradycardia, which likewise occasionally occurs in attacks, see above, page 417.

SECTION II

Diseases of the Pericardium

CHAPTER I

PERICARDITIS

(Inflammation of the Pericardium)

ÆTIOLOGY

Pericarditis seldom appears as a primary idiopathic disease. It is usually merely a sequel or a complication of other diseases. Thus, it is observed with particular frequency in the course of acute articular rheumatism, where it appears sometimes alone, sometimes in combination with acute endocarditis.

It is not impossible that some few cases of apparently primary acute pericarditis belong, from an ætiological standpoint, to acute articular rheumatism—that is, they are excited by the same pathogenic factors which exceptionally attack the pericardium alone, without simultaneous participation of the joints in the disease. This supposition is rendered probable from the later course of many such cases. For instance, arthritis may ensue. Cases of secondary pericarditis, unassociated with articular rheumatism, occur, although much less often, in other acute infectious diseases, among which scarlet fever, measles, and septico-pyæmic processes, as well as scurvy and purpura hemorrhagica, deserve especial mention. In sepsis and pyæmia the pericarditis is purulent, and in purpura, hemorrhagic. Among the chronic diseases in the course of which pericarditis sometimes appears we must mention especially chronic nephritis. We have seen hemorrhagic pericarditis in association with leukæmia. Pericarditis sometimes occurs also in the victims of carcinoma, but here it is probably dependent upon a secondary septic infection. Finally, it should be mentioned that severe hemorrhagic pericarditis is seen comparatively often in alcoholic subjects, and apparently as a primary disease. Often it is really tubercular, but by no means always; and if not, the author is inclined to regard it as a primary hemorrhagic inflammation, analogous to hemorrhagic pachymeningitis (hematoma of the dura mater).

A large number of cases arise from an extension of the inflammation from the vicinity. Thus we not infrequently see pericarditis as a result of pleurisy, especially on the left side, and in pneumonia complicated with pleurisy. New growths and ulcerative processes in the esophagus, in the vertebræ, in the bronchial glands, or in the lungs, also lead at times to perforation into the pericardium and a consequent inflammation. It is not settled whether the pericarditis which appears in the course of chronic valvular disease is also to be regarded as arising from extension by contiguity. We have thought of this possibility, because we have been struck with the fact that secondary pericarditis is especially frequent in valvular disease of the aorta, suggesting a direct propagation of the inflammatory germs through the aortic wall to the pericardium. Still, it must, of course, be allowed that this form of pericarditis may have an independent origin, particularly in cases of mitral disease. Pericarditis may also develop as a result of myocarditis, abscess of the heart, etc.

Tuberculosis plays a very important part in the ætiology of pericarditis. No small number of apparently primary cases of pericarditis turn out at the autopsy to be tuberculous. This seems to come on in quite an isolated way, or as one symptom of a special localized form of tuberculosis, which we term tuberculosis of the serous membranes. In many we can discover the origin of a tuberculous pericarditis in the direct extension of a tuberculous pleurisy. In apparently primary cases the occurrence of the infection may sometimes be explained by the discovery of a tuberculous lymph-gland, which has broken through into the pericardium.

Pericarditis is usually a disease of youth and middle life, but it may also occur in advanced age.

PATHOLOGICAL ANATOMY

Ordinary pericarditis involves the internal surface of the pericardium in either a circumscribed or diffuse manner. Inflammation of the outer sur-

face of the pericardial sac is distinguished as external pericarditis (*vide infra*). The anatomical processes in pericarditis are precisely analogous to those in inflammations of the serous membranes in general, especially of the pleura.

We usually divide pericarditis into fibrinous, serofibrinous, hemorrhagic, and purulent (or ichorous) forms, according to the character of the exudation. The fibrinous and serofibrinous forms, with an abundant fluid effusion into the pericardial cavity, are the most frequent, occurring in articular rheumatism, in valvular disease of the heart, etc. Both layers of the pericardium are covered with masses of fibrin, which often show a reticular or villous arrangement (*cor villosum*). Besides this, we find more or less of a fluid effusion which distends the pericardium. The fluid is of a serous nature, contains more or less numerous flakes of fibrin, and is turbid from the admixture of cells—pus corpuscles, and, in part, desquamated endothelium. A purulent pericarditis is always the expression of a specific infection of the pericardium. It is seen in pyæmic diseases, as a result of empyema, and in perforation of abscesses, cancers of the œsophagus, etc., into the pericardium. A hemorrhagic effusion is seen chiefly in tuberculous pericarditis. In this we find miliary tubercles, and little cheesy nodules in the inflammatory new growths, besides all the signs of inflammation. The specific tuberculous changes are sometimes recognizable with the naked eye, but at other times we have to use the microscope to find them. Hemorrhagic pericarditis also occurs in general hemorrhagic diseases, such as scurvy, and in weak and debilitated persons, especially drunkards (*vide supra*).

In long-continued pericarditis the cardiac muscle almost invariably undergoes changes. The heart is usually flabby and dilated, and the muscle often shows fatty degeneration. After the pericarditis has lasted a long time there is often considerable atrophy of the cardiac muscle, which is partly replaced by fat tissue. We have already mentioned the occurrence of pericarditis in connection with valvular disease and degenerations of the myocardium.

In favorable cases of pericarditis we may have a perfect recovery. The so-called *maculæ tendineæ* sometimes remain in the pericardium as residua of a past circumscribed pericarditis. In some cases the pericarditis leads to an adhesion of the two layers of the pericardium to each other, and obliteration of the pericardial cavity (*vide infra*). In many cases a chronic pericarditis finally develops from the acute form, or the whole affection takes a more chronic course from the outset. In this way chronic adhesions of connective tissue arise, and great thickening of the pericardium, but the amount of fluid is usually small. Sometimes the chronic pericarditis is interrupted by an acute exacerbation of the disease.

CLINICAL SYMPTOMS

1. Subjective Symptoms, General Symptoms, and Fever.—Mild forms of pericarditis may develop, as in the course of an acute articular rheumatism, without causing any subjective symptoms. They are discovered only by a careful physical examination of the heart. In severe cases, however, the pericarditis causes violent subjective symptoms, which, of course, have in themselves little that is characteristic.

On careful questioning we often find that there is pain in the cardiac re-

gion, or not infrequently in the epigastrium. This pain is of decided diagnostic importance, but it does not always exist.

A general feeling of constraint and distress is almost constant in all acute cases of any severity, and so is a feeling of dyspnœa, which may increase to the highest degree of orthopnœa. The patients often complain of headache. In severe cases they become stupid and comatose.

It is easy to understand that extensive pericarditis must impede the motions of the heart. Probably the most important way in which it does this is by increasing the tension in the pericardium, and thus embarrassing the diastole of the ventricles, so that the cavities of the heart are not normally filled and general disturbance of the circulation ensues. Furthermore, the pericarditic deposits and adhesions, and such changes in the myocardium as may occur, interfere with the cardiac systole. Thus are explained the early signs of diminution in the amount of blood received by the arteries, as shown by pallor and cerebral anæmia; the distention of the systemic veins, as shown by cyanosis; and the disturbance of the pulmonary circulation, causing dyspnœa.

The dyspnœa is also increased in large pericardial effusions by the mechanical pressure of the distended pericardium on the left lung.

Acute pericarditis is usually associated with fever. This has no special type, and usually keeps at a moderate height—102° to 103.5° F. (39° to 39.8° C.)—but it often exhibits considerable variations. In cases of recovery the fever declines by lysis. Chronic pericarditis may run its whole course without fever.

2. **Physical Signs.**—*Inspection.*—The general hue of a patient with severe pericarditis is pale, but also more or less cyanotic. He has an anxious expression. He lies with the upper part of the body raised, or he sits up in bed. The breathing is usually rapid, labored, and somewhat irregular. The veins in the neck are swollen and prominent. We very often see marked undulating or pulsating movements in the jugular veins, as a result of stasis. The cardiac region seems unusually prominent in all cases with much effusion, and the intercostal spaces there are flattened out. We sometimes detect a slight œdematous swelling of the chest wall itself.

If the action of the heart is powerful and the exudation small, the heart's movements may be distinctly visible. In other cases they are seen only faintly, and they may be noticeably diffuse. If the exudation is abundant, or if adhesions occur (*vide infra*), they may disappear altogether.

Palpation.—Palpation in the milder cases shows the apex beat in its normal position and of about normal strength; but if the amount of the pericardial effusion increases, the heart is pushed away from the chest wall by it, and hence the heart beat grows weaker until it disappears entirely. In such cases it is sometimes to be felt again if the patient bends forward or lies on his left side. In the rest of the cardiac region we sometimes feel the movements feebly, but they entirely disappear as the effusion increases. There is diagnostic importance in the contrast between the great extent of cardiac dullness (*vide infra*) and the faintly distinguishable motions of the heart, with absence of a distinct apex beat and any marked epigastric pulsation. In some cases, by laying the hand flat on the chest, we can feel the rub of the rough pericardial surfaces against each other.

The pulse is usually accelerated, and in severe cases it becomes irregular. In every large effusion, as we have already said, the tension and height of the pulse are diminished. In severe cases the pulse sometimes becomes very small and weak, but, when the heart is otherwise normal and strong, it may also remain quite strong—and indeed this condition of the pulse, in contrast to the great weakening of the heart beat, is sometimes of diagnostic significance. In some cases with a large pericardial effusion we have seen a manifest *pulsus paradoxus*—that is, a diminution or a complete disappearance of the radial pulse on every inspiration.

Percussion.—Changes in percussion resonance are evident as soon as the pericardial sac is distended with exudation. The so-called triangular or bottle shape of cardiac dullness is regarded as especially characteristic of large pericardial exudations. The blunt apex of the triangle is found above in the second or third left intercostal space, near the edge of the sternum. The lateral boundaries run obliquely to the right and downward to about the right parasternal line, and to the left and downward to the left mammillary line, or beyond. The broad base of the triangle which lies below is usually not to be defined by percussion, on account of the adjacent left lobe of the liver. On the border of the dullness we often find a tympanitic resonance due to the retraction of the adjacent lung.

In general, the author's personal experience forces him to say that we must not be too dogmatic in the establishment of special shapes of cardiac dullness, as peculiar to pericarditis. The fact of enlargement of the area of cardiac dullness, and the marked sense of resistance upon percussion, are important signs, but there is a considerable variety in the shapes of the dull area, although extension of the dullness upward and to the right may be regarded as particularly frequent. According to Ebstein's observations, the first change in percussion in incipient exudation is usually an extension to the right of the normal cardiac dullness, in a triangle between the heart and the liver.

The area of the dullness depends, of course, in the first place, upon the amount of the effusion, but we must take special notice that in regard to this the relation is not constant. In old cases of pericarditis especially we sometimes find the cardiac dullness quite extensive, while the autopsy detects only a little fluid in the pericardium. This is explained partly by a secondary dilatation of the heart, and partly by a persistent retraction of the lung.

It is an often-mentioned but seldom available diagnostic sign of pericarditis that in many cases the still perceptible apex beat lies within the cardiac dullness, since the pericardial effusion extends farther to the left than the heart itself. It is also worthy of note that the dullness in pericarditis often shows very great changes when the patient changes his position. The dullness is more extensive when the body is erect than when lying down, and when the patient lies on his side it sometimes shows a lateral displacement of several centimeters. The same changes, however, though rarely so marked, also occur in an hypertrophied heart.

Auscultation.—The characteristic pathognomonic auscultatory sign of pericarditis is the pericardial friction rub. This arises during the movements of the heart from the rubbing of the rough and inflamed pericardial surfaces against each other. The friction rub is absent in pericarditis if the rough surfaces of the two layers of the pericardium are separated from each other

by a considerable fluid effusion, or if they can no longer rub against each other from an adhesion of the layers of the pericardium. We usually hear the friction rub loudest in the neighborhood of the base of the heart, but it may also be heard at other parts of the heart. In general the adventitious sounds of pericarditis are not transmitted to any great distance. The quality of the sound is described as rubbing, grating, or scratching. The friction rub may be heard chiefly either during the systole or during the diastole of the heart, but it is in general not often closely associated with the phases of the heart's action. We sometimes find it intermitting frequently, and jerky. The intensity of the friction rub sometimes varies with the phases of the respiration. It is usually louder on inspiration, but sometimes on expiration. If the patient changes his position, the intensity of the sound is sometimes altered. It is louder when sitting up than lying down, etc. The friction rub often sounds louder if the stethoscope is pressed firmly against the chest, since in this way the layers of the pericardium are approximated to each other.

The heart sounds, when the valves are intact, may sometimes be heard as well as the friction rub, or they may be completely drowned by the loud rub, at least in some parts of the heart. In general, they are faint in every case of pericardial effusion, since their conduction to the ear is impaired. In large effusions where no friction rub is to be heard, we hear the heart sounds, especially the first, but only very faintly and obscurely. This condition, in connection with the increase of the cardiac dullness, is of diagnostic importance. If there is also valvular disease with the pericarditis, the pericardial and endocardial murmurs are sometimes hard to distinguish from each other, but usually the former greatly preponderate.

3. **Sequelæ of Pericarditis.**—A large pericardial effusion may excite special symptoms from pressure on the neighboring organs. Thus we have already said that compression of the left lung must increase the dyspnœa. In many cases we also notice a moderate dullness over the left lower back, from compression of the left lower lobe. Not infrequently there is a combination of pericarditis and left-sided pleurisy with effusion. In rare cases difficulty in deglutition has been observed as a result of pressure on the esophagus, and paralysis of one vocal cord from pressure on the recurrent nerve.

In cases of long-continued pericarditis the same sequelæ may develop as in any chronic disease of the heart. The amount of urine diminishes as a result of the low arterial pressure. The venous stasis finally leads to general dropsy and to symptoms of passive congestion in the liver, spleen, and kidneys. We would also state that we have repeatedly met with large effusions in the cavities of the body, especially hydrothorax, without any œdema of the skin. All the symptoms of stasis mentioned, however, are often due much less to the pericarditis itself than to the atrophy and dilatation of the heart which frequently follow it (*vide supra*).

SPECIAL FORMS OF PERICARDITIS

1. **Pericarditis externa and Mediastino-pericarditis** (*Pleuro-pericarditis*).—By pericarditis externa we mean an inflammation of the external surface of the pericardial sac, which is usually combined with an inflammation of the mediastinal connective tissue and the neighboring pleura, especially over the

lingula of the left lung. This form of pericarditis may exist by itself, or it may be combined with internal pericarditis. It is a rare disease, and is most frequently seen as a result of tuberculous pleurisy.

The physical signs must differ so much, according to the localization and extent of the process, that we can give few general data in regard to them. There are only a few peculiar signs, which must be noted as characteristic of many cases. In the vicinity of the apex beat, or at the left border of the cardiac dullness, we sometimes hear a so-called extra-pericardial (pleuro-pericardial) friction rub. This depends both upon the cardiac movements and upon the respiratory movements. The friction sound often becomes louder during inspiration, or, again, it may be audible only when the lungs are fully inflated. On holding the breath we hear merely the sound due to the pulsations of the heart, while on deep breathing the pleuritic friction sound is also to be heard. In individual cases there are many modifications, which cannot all be mentioned. Another interesting sign, first found by Griesinger and Kussmaul in a cicatricial mediastino-pericarditis, is the so-called *pulsus paradoxus*. This consists of a diminution of the pulse at each inspiration. This condition arises, in part of the cases at least, from the fact that the bands and adhesions of connective tissue at the origin of the aorta mechanically nick into and contract its lumen at every inspiratory movement of the thorax. This explanation, of course, does not suffice for all cases, since the *pulsus paradoxus* also occurs under other conditions, as with large pericardial effusions. In some cases there may be seen a marked swelling of the jugular veins in the neck at each inspiration, at the same time with the *pulsus paradoxus,* since the large venous trunks also undergo a mechanical nicking and constriction at each inspiration. We have ourselves seen a very pronounced slowing of the pulse at every inspiration, in a complicated case of extra-pericardial adhesions (vagus irritation?). We must also mention that Riegel observed a disappearance of the apex beat on expiration in some cases where there were bands of connective tissue between the lungs and the outer surface of the heart. At every expiration the bands were stretched more tightly, and hence checked the movements of the heart.

2. **Obliteration of the Pericardial Cavity** (*Adhesive Pericarditis; Adhesions of the Layers of the Pericardium; Concretio seu Synechia pericardii*).—We may have a more or less complete adhesion of the two layers of the pericardium with each other as a result of pericarditis. We can sometimes observe the occurrence of this condition during the course of a pericarditis. Quite frequently, however, we meet with extensive adhesions of the two layers of the pericardium at autopsies, without being able to gather any history of a previous acute pericarditis. The pericarditis must have occurred here in a chronic way, and without symptoms from the outset.

Even extensive adhesions of the pericardial surfaces may develop and remain entirely without symptoms, and be met with accidentally at the autopsy. In other cases, however, the obliteration of the pericardial sac causes special physical signs and severe clinical sequelæ. Of the physical signs, one of the best known and most discussed is the systolic retraction of the chest, either limited to the apex or involving a larger area. This is most comprehensible if there is an adhesion of the pericardium with the heart, and also with the chest wall (Skoda); but we certainly find this retraction at the systole with-

out coexisting extra-pericardial adhesions. It is not, however, an absolutely certain sign of an intra-pericardial adhesion, especially if we have to do with a systolic retraction at the apex alone, or limited to the third or fourth intercostal space, since systolic retractions may sometimes occur in other disturbances of the heart's motions, or even under normal conditions; but systolic retractions of the whole cardiac region are, in the majority of cases, a certain sign of pericardial adhesion. The amount of this retraction is often dependent upon the respiration, it being usually more marked on inspiration.

On the other hand, it must be noted that very often there is no retraction of the præcordia during systole in many cases of undoubted obliterative pericarditis. In brief, the symptom under consideration must always be viewed with considerable reserve in forming a diagnosis. Of at least equal importance, in our opinion, is the obscurity or absence of any cardiac motion either to sight or touch. We can lay great diagnostic value upon the absence of a cardiac impulse when the cardiac dullness is increased in breadth and upward, although the heart sounds are normal; and when the adjacent borders of the lung do not move with respiration, because of the adhesions due to the associated pleurisy (*vide infra*); and finally, when the history of the case and the general course of the illness are confirmatory, for instance, if there are indications of a previous acute pericarditis.

The other symptoms of obliteration of the pericardial cavity are more rare and in their diagnostic significance still more uncertain. Friedreich observed a sudden collapse of the jugular veins at each diastole—the "diastolic collapse"—while they became well filled again at the next systole. He explained this phenomenon by supposing that the conditions for emptying the veins at the moment of the diastole of the ventricle were especially favorable, since the chest wall, which had previously been drawn in by the systole, sprung back again quickly. But we have ourselves seen distinct diastolic collapse of the veins in a case of adhesive pericarditis, the diagnosis of which was confirmed by autopsy, in which there was no systolic retraction of the præcordia. Reiss described some cases of pericardial adhesions in which the heart sounds had a metallic character from the resonance of the stomach, which had been drawn up. All things considered, we must say that although the diagnosis of pericardial adhesions can be correctly made in many cases, yet the signs given for it are more or less uncertain, since they may be absent in obliteration of the pericardial sac, and they may also be caused by other conditions without such an obliteration. In a word, it is no easy matter to establish the diagnosis of obliterative pericarditis, and in cases of this sort the autopsy not infrequently brings surprises, as our own experience has shown.

The general constitutional symptoms of obliterative pericarditis are also subject to great variations. In many cases the affection, as we have said, has no symptoms at all or at least no grave results. In other cases, however, there is distinct evidence of circulatory disturbance. This disturbance is sometimes due directly to the mechanical embarrassment of the heart, but more often to secondary changes in the myocardium. We fine atrophy, fatty infiltration, and fatty degeneration, with consequent dilatation. In such cases the pulse becomes small and frequent, and the ordinary picture of ruptured compensation is displayed, characterized by œdema, dyspnœa, and congestion of the liver and kidneys. In these cases the diagnosis is often very

difficult. It is scarcely possible to distinguish between this form of pericarditis and chronic myocarditis, or the "idiopathic" enlargement of the heart.

There is another symptom-complex, somewhat different from the above, which deserves special mention. We have been acquainted with it for years, but it has only lately received general attention. Sometimes, when there is obliteration of the pericardial sac, there is developed a condition so like that of hepatic cirrhosis (*q. v.*), that Friedel Pick has suggested the name of "pericarditic pseudo-cirrhosis of the liver" for it. There are increasing dyspnœa and great ascites, with subsequent œdema of the lower extremities, so that we have dropsy of the lower half of the body, while the upper half remains free from œdema (see Fig. 65). The veins of the neck, however, are usually markedly distended, the face is cyanotic, and not infrequently a serous effusion is demonstrable in one or both pleural cavities. At the autopsy we do not find the hepatic cirrhosis which has in many cases been supposed to exist, but instead, a total obliteration of the pericardial sac, with which is often associated a nutmeg liver, and often, also, chronic peritonitis and chronic pleurisy. The peritonitis causes a marked fibrous thickening of the peritoneum, apt to be especially marked on the surface of the liver, in the form of what is called "frosted liver." This condition, therefore, in many cases, should be classed as one of "chronic inflammation of the serous membranes" (*vide supra,* page 352). We may have a similar group of symptoms not associated with inflammation of the peritoneum, if the circulatory disturbance resulting from the obliteration of the pericardium affects chiefly the portal circulation. We have also observed a combination of obliterative pericarditis with genuine hepatic cirrhosis. In a word, the cases are not all after one pattern. The diagnosis can often be correctly made when one is acquainted with the symptom-complex, and pays especial attention not only to the condition of the other serous cavities, and to the history of the case, but to the cardiac signs, such as increase in the area of dullness, absence of apex beat, or presence of systolic retraction.

3. **Tuberculous Pericarditis.**—Tuberculous pericarditis is an important disease clinically, since in many cases it is apparently primary. It may be either quite acute or chronic. The patient falls ill suddenly, or more gradually, with indefinite thoracic symptoms, dyspnœa, general weakness, moderate fever, etc. If the disease is of long duration, there is more or less œdema. When we find on physical examination, in such cases, the signs of pericarditis, the diagnosis of tuberculous pericarditis is probable, if we discover a general "phthisical habit," hereditary predisposition, and also coexisting disease of other serous membranes, especially pleurisy, or more rarely chronic peritonitis. In the latter case the tuberculous pericarditis forms one symptom of the so-called tuberculosis of the serous membranes, but, as has been said before, apparently isolated primary tuberculous pericarditis does occur (*vide supra*). We have seen such cases repeatedly, especially in old people. In these cases the disease is not easy to diagnosticate. The patient gives one the impression of having heart disease, but the physical signs in the heart are sometimes of a very indefinite nature. Friction rubs may be entirely absent, on account of adhesions or of large effusions. This leads to confusion with myocarditis or mitral stenosis. In other cases, of course, all the physical signs of peri-

carditis mentioned above may be manifest, and a correct diagnosis can be made.

DIAGNOSIS

From what precedes, it follows that the diagnosis of pericarditis is very easy in many cases, but is very difficult or impossible in others. The most unequivocal sign is the characteristic friction rub. The practiced ear can

FIG. 65.—Pseudo-hepatic cirrhosis due to pericarditis. (Personal observation.)

often distinguish it from an endocardial sound by its quality. The pericardial sound is a rubbing, grating noise, near the ear; the endocardial is blowing distant from the ear. The following features may serve as marks of distinction in doubtful cases: 1. We hear the pericardial sounds at first, and also later, over the base of the heart in the vicinity of the pulmonary valve; the endocardial are often loudest at the apex. 2. The pericardial murmurs are

not so closely associated with the phases of the heart's action, with systole and diastole, as the endocardial. 3. We find that the pericardial sounds are not transmitted far. A loud rub may be audible at one spot which cannot be heard a few centimeters away. Loud endocardial murmurs, however, are audible over almost the whole heart. 4. Sometimes the peculiarity of the pericardial murmur—that it becomes louder when the patient sits up, on pressure with the stethoscope, etc.—may be of diagnostic value. In many cases the loud, functional, so-called anæmic murmurs over the base of the heart may give rise to confusion with pericarditis.

If no pericarditic friction has been heard during any part of the illness, it is seldom possible to make an absolute diagnosis of pericarditis. At least, we must confess that we have made many errors in the diagnosis of such cases. Important factors to be considered are the general course of the disease, including the acute onset and præcordial pain, and also the shape of the cardiac dullness (triangular), and the signs furnished by the apex beat, the pulse, and the heart sounds. We have already pointed out the possibility of confusion between pericarditis and myopathic disease of the heart, and mitral stenosis if unattended by murmur. No rules of universal application in these cases can be laid down. It is very often advisable, and absolutely devoid of danger, to make an exploratory puncture in doubtful cases with a hypodermic syringe. This is done at the left edge of the sternum, or a little way in from the left limit of cardiac dullness.

COURSE AND PROGNOSIS

Many cases of pericarditis in articular rheumatism, pneumonia, or heart disease, and also many of the rare and apparently primary forms, may recover completely. In rheumatic pericarditis I have usually seen recovery even in apparently the severest cases. The disease lasts, in the mild cases, only about a week, in severe cases much longer.

Many cases of pericarditis, however, terminate fatally. The unfavorable issue depends either upon the severity of the primary disease, or upon the intensity of the pericarditis itself. In extensive croupous pneumonia, in valvular disease of the heart, or in severe chronic nephritis, an attack of pericarditis is often the terminal affection—the immediate cause of death. In otherwise healthy persons, however, a severe pericarditis with a large effusion may be the direct cause of death, as a result of the impairment of the movements of the heart. Yet one need not wholly despair, particularly in rheumatic pericarditis, even in cases which seem very bad. The prognosis of tubercular pericarditis is always very unfavorable. This form may, indeed, run quite a chronic course, but it is hardly ever capable of definite recovery. The prognosis of pyæmic pericarditis is also unfavorable.

In one class of cases pericarditis takes a chronic course from the start, or chronic pericarditis develops from an acute attack. The ultimate prognosis of these cases is usually unfavorable, since the secondary atrophy and dilatation of the heart gradually lead to severe disturbances of the circulation. We have spoken above of the termination of pericarditis in obliteration of the pericardial sac.

TREATMENT

Since pericarditis is a severe affection under all circumstances, we must see especially that the patient has perfect rest and care. Extreme caution must be enjoined upon him, especially in the cases in which at first the subjective symptoms are slight. We must keep the patient strictly confined to the bed, and not let him leave it even temporarily.

The remedies which are used for pericarditis aim partly at keeping the inflammation in check, and partly at aiding the action of the heart. For the first, the continued application of ice to the cardiac region deserves especially to be recommended. Local bloodletting, ten or twelve leeches to the cardiac region—formerly very often, but now more rarely used—may, in otherwise strong and healthy persons, afford great relief in cases with marked subjective symptoms. Painting with tincture of iodin and blistering, however, deserve little confidence. Digitalis is our chief means to bring down an accelerated pulse and to strengthen the heart's action. It is the drug which is the most efficient and the most frequently used in pericarditis, and is always indicated when the pulse is rapid and of diminished tension. Of course, the action of the remedy must be carefully watched, as in all cases where digitalis is prescribed. Caffein and tincture of strophanthus are also useful. As a palliative, morphin often does indispensable service when the subjective symptoms are marked and the patient is very restless. Diuretics are also employed, as in pleurisy with effusion (*q. v.*)

If the symptoms are threatening, the question arises whether a large fluid pericardial effusion is the cause of the severe symptoms. In this case the evacuation of the exudation is, of course, imperatively indicated. The difficulty of forming a correct opinion, however, is very great, because in any individual case it is rarely possible to determine the amount of fluid that may be present. In the first place, we must consider the size of the cardiac dullness and the weakening of the movements of the heart, but both factors may give rise to deception. Hence we always first make an exploratory puncture with a Pravaz's hypodermic syringe. According to the newer extended experiences of Curschmann, the best point of insertion is in the left mammary line, or, if the exudate is large, a point even more or less external to this, somewhat internal to the outermost limits of the area of dullness. If the height of the diaphragm is normal, the fifth intercostal space is entered, while if this is low, the sixth is utilized. The exudate is siphoned off with the aid of a Fiedler needle or a sheath-shaped Curschmann trocar. Of course, in certain cases, other points of puncture must be selected. With regard to the details, we will refer to the description of puncture of the pleura. Puncture of the pericardium is always performed by the aid of aspiration. It is less dangerous than might be feared. Even injuries to the heart during the operation have scarcely ever had grave results. The temporary relief to the patient, in cases of successful puncture, is usually very striking, but the permanent results of pericardial puncture are, of course, much less favorable than those of puncture of the pleura, which is chiefly due to the character of the underlying disease. In some cases of purulent pericarditis, incision of the pericardium has also been practiced after the analogy of the treatment of empyema. In other forms of pericarditis with an abundant fibrino-serous

exudation, it might often be preferable to make an incision rather than to aspirate, but there has not yet been much experience with the former method.

If there is a condition of cardiac weakness, stimulants are indicated—strong wine, subcutaneous injections of caffein or camphor, strophanthus, etc. We try to keep up the patient's strength by the best of nourishment.

The resulting conditions of disturbance of the circulation, such as œdema, in chronic pericarditis, are treated in the same way as in valvular disease (*vide supra*). Digitalis and diuretics are the chief remedies.

In obliteration of the pericardium, Brauer has recommended a surgical procedure, the so-called cardiolysis—i. e., the resection of the ribs in the præcordial region. His idea is thereby to obtain greater freedom and facility for the cardiac contractions. The operation has already been frequently performed, with a fair percentage of really favorable results, especially, as I have personally been able to observe, in cases of so-called pericarditic pseudocirrhosis of the liver.

CHAPTER II

HYDRO-PERICARDIUM, HEMO-PERICARDIUM, AND PNEUMO-PERICARDIUM

1. HYDRO-PERICARDIUM

(Dropsy of the Pericardium)

The collection of a serous transudation in the pericardial sac, without any inflammatory symptoms in the serous membrane itself, we term hydro-pericardium, or dropsy of the pericardium. Dropsy of the pericardium, which formerly played quite a great rôle in pathology, is never a disease of itself, but is always a secondary condition. It may occur in anæmic and cachectic persons as a result of hydræmia, but it usually depends upon a local or general venous stasis in the pericardium. In the latter case the hydro-pericardium is one symptom of general dropsy, and hence is found chiefly in heart disease, renal disease, or pulmonary emphysema.

The clinical symptoms of hydro-pericardium are only exceptionally distinct, being obscured by the underlying affection. Large amounts of fluid in the pericardial sac, which may amount to a quart (a liter) or more, must, of course, impair the action of the heart, weaken the heart beat objectively, and cause an increase in the cardiac dullness. Nevertheless, if the lungs are markedly emphysematous, even large pericardial transudates are easily overlooked. The distinction from pericarditis is rendered possible by the absence of a friction rub, but still more by attention to the existence of an underlying disease. Notwithstanding this, errors are easily possible.

The prognosis and treatment depend wholly upon the nature of the underlying disease. Only exceptionally do we need to puncture when the exudation is very large.

2. HEMO-PERICARDIUM

(*Blood in the Pericardial Sac*)

In rare cases hemorrhages occur into the pericardial sac. The source of the hemorrhage is most frequently an aneurism of the aorta, which perforates into the pericardium. Other causes of hemorrhage are the bursting of aneurisms of the coronary arteries and rupture of the heart. The latter has been seen after injuries, and also as a result of cardiac aneurism and the cicatricial formations in myocarditis (see myocarditis). Finally, direct injuries to the heart, especially bullet wounds, may also cause hemorrhages into the pericardial sac.

In most cases death occurs in a few moments from compression of the heart, when a hemo-pericardium comes on. Hence the amount of blood poured out into the pericardial sac is usually not very considerable. Only in the cases in which the blood oozes out more slowly can a great distention of the pericardial sac be reached. The diagnosis is rarely possible. With regard to treatment we can merely note that, in some traumatic cases, aspiration of the blood has been performed with success.

3. PNEUMO-PERICARDIUM

(*Air in the Pericardial Sac*)

The entrance of air or gas into the pericardial sac has been observed in rare cases, apart from external wounds, as a result of the perforation of a pyo-pneumothorax, or of some other suppurating process in organs that contain air. Thus, cases are known in which the rupture into the pericardial sac comes from the œsophagus, as in cancer; from the stomach, in cancer or ulcer; or from the lungs, in tubercular or gangrenous cavities. Since the agents of inflammation enter the pericardium along with the air, a purulent pericarditis almost always develops, besides the pneumo-pericardium, or it may rarely be simply a sero-fibrinous pericarditis.

The most characteristic and striking sign of pneumo-pericardium is the presence of a metallic sound, due to the movements of the heart. Either the heart sounds themselves, or some existing friction rub, may acquire a metallic timbre from the increased resonance, or splashing metallic sounds may be produced in the pericardial sac from the movements of the air and the fluid, which may often be heard at a distance from the patient. In regard to diagnosis, however, it is important to know that signs similar to those of metallic resonance in the heart may arise from the stomach, when it is drawn or pushed upward.

In true pneumo-pericardium percussion gives a more or less complete absence of the cardiac dullness. On rod percussion (see page 362) a metallic sound is sometimes heard, whose pitch may vary somewhat with the phase of the heart's action. If fluid is also present in the pericardial sac besides the air, the dullness caused by this will rise on raising up the patient.

The other symptoms of the disease and the treatment are the same as in a severe pericarditis. The prognosis, however, corresponding to the primary disease, is wholly unfavorable.

SECTION III

Diseases of the Vessels

CHAPTER I

ARTERIOSCLEROSIS

(Endarteritis chronica deformans. Atheroma of the Vessels)

Ætiology.—Arteriosclerosis, or so-called atheromatous degeneration, is probably in most cases an expression of the wearing out of the arteries, because of the many ways in which the intima of the arteries is damaged during life, both chemically and mechanically. Atheroma of the arteries is then mainly a change incident to age, and as such is often regarded as not a true disease.

On the other hand, if arteriosclerosis appears in earlier life, say before the fortieth year, we must look for such circumstances as would tend to make the ordinarily efficient causes unusually active. We must not forget that there is an apparent predisposition to the condition, which often seems to be hereditary; but without doubt there are more appreciable causes. The reason that arteriosclerosis is so common and occurs comparatively so early in hard laborers, is the great mechanical strain which the arteries undergo during persistent, severe, bodily exertion. Again, in diseases which are associated with a persistent elevation of arterial tension, such as hypertrophy of the left ventricle in interstitial nephritis, arteriosclerosis develops probably as a result of the constant high tension. It must be confessed that here a decision is often very difficult, inasmuch as the arterial disease may be simultaneous with the nephritis, or even the actual cause of it (arteriosclerotic contracted kidney). Again, excessive chemical irritation must be considered. It is of great theoretical interest that by repeated adrenalin injections a sclerosis very similar to the arterial disease can be artificially produced in rabbits (Jousé, Erb, Jr., etc.). Experience shows that habitual excess in eating and drinking, particularly in the use of alcohol, is apt to occasion arteriosclerosis, and in the same connection are to be named such constitutional diseases as gout and chronic nephritis, and perhaps also definite poisons such as lead. Of late, particular importance has been correctly attributed to continuous excessive smoking (chronic nicotin poisoning and other harmful factors). From personal experience, I can substantiate the frequent early development of arteriosclerosis in heavy smokers. Nor can we disregard the possibility that the disease may follow persistent mental strain and excitement. All these circumstances explain why well-marked atheroma is far more frequent in men than in women. Syphilis plays a peculiar rôle in the ætiology of arteriosclerosis. We know now that many cases of sclerosis of the aorta, as well as of the other arteries, are actual syphilitic lesions.

Pathological Anatomy.—Atheroma is almost exclusively confined to the arteries; only exceptionally do like processes occur in the veins. Among the arteries the aorta is almost always the most intensely and extensively diseased;

we also find disease in the iliac and femoral arteries, the brachial, radial, and ulnar, the coronary arteries of the heart, and the arteries of the brain. In some of the other arteries, however, like the gastric artery, the hepatic, and the mesenteric, we very rarely find atheromatous changes.

Nevertheless, in the individual cases, great variety is found in regard to the principal localization of the arteriosclerosis. Sclerosis of the peripheral vessels is not always associated with marked aortic sclerosis, and *vice versa*. Sometimes the sclerosis is especially well developed in the cerebral vessels.

The atheromatous process is easy to recognize macroscopically. Instead of the normal smooth internal surface, we find more or less numerous irregularities and thickenings on the intima, which appear either more or less gelatinous and translucent, or dense and fibrous, or ossified as a result of calcification, in which case they also feel perfectly hard. In many cases we find the surface of the thickenings destroyed—atheromatous ulcers—and covered with masses of fibrin.

The wall of the arteries is usually increased in thickness, making the vessel feel stiff and hard. Very frequently the calcareous plates may be felt by the finger. The frequently associated increase in arterial tension gradually stretches the walls of the arteries, rendering them tortuous. This is best seen in those of medium size, such as the brachial and radial.

Microscopic examination shows that the chief changes are situated in the intima of the arteries. This appears three or four times as thick as normal, partly from the swelling of its elements and partly from the new growth of connective tissue and the deposit of round cells. In the connective-tissue cells of the intima, and in the endothelial cells of its surface, we usually find a marked fatty and hyaline degeneration, to which the yellowish, translucent appearance of the surface is due. Finally, in the deeper layers there is a complete breaking down of the tissue into a mixture of fat, detritus, and cholesterin crystals, which has given the whole process the name of atheroma [= pulp]. If this destruction extends to the surface, an atheromatous ulcer is formed. In other places, however, it does not reach ulceration, but the superficial layers of the intima become sclerosed, and are finally changed to lamellæ of bony hardness from the deposition of lime salts. The atheromatous spots on the intima of the vessels often give rise to the formation of parietal thrombi.

The media and adventitia of the arteries also show changes, in the later stages of the process. Here, too, we may finally get fatty degeneration and calcification. In other cases, however, there is a marked atrophy of the media.

The immediate result of the atheromatous changes is a loss of elasticity in the walls of the vessels. The ability to resist the blood pressure is reduced, and this is why diffuse or circumscribed aneurismal dilatations of the vessels so often arise as a result of arteriosclerosis (see the following chapters).

Another result of extensive atheromatous degeneration of the vessels is an increase of the resistance of the blood current and a consequent elevation of the arterial pressure. Furthermore, the loss of elasticity in the coats of the medium-sized and smaller arteries removes an important factor for the propulsion of the blood. The left ventricle, in consequence of the additions to its task, becomes almost invariably hypertrophied in cases of extensive arteriosclerosis, provided the general nutrition of the patient is still well maintained.

We see, therefore, how close the mutual relations are between increased arterial tension, hypertrophy of the left side of the heart, and arteriosclerosis.

The thickening of the intima in the smaller vessels often causes so marked a diminution of the blood supply that secondary disturbances of nutrition are not wanting in the various organs. The lumina of the vessels may be still further narrowed, or even completely closed, by the formation of thrombi on such portions of the wall of the vessels as have undergone atheromatous changes. We have already in part learned to recognize the sequelæ which necessarily arise in the various organs, such as indurations in the heart as a result of atheroma of the coronary arteries, and we will return later on to the analogous changes in some other organs, such as cerebral softening and certain forms of contracted kidney.

Clinical Symptoms.—In order to decide whether an arteriosclerosis is present in the living subject, we are, of course, restricted exclusively to the examination of those peripheral arteries which are accessible to palpation. We must examine, first of all, the radial, brachial, femoral, and temporal arteries. If there is atheroma, we feel the hard and partly calcified vascular tube. In marked cases we have a feeling, especially in the radial, as if we had hold of a goose's neck. We sometimes notice a diffuse dilatation of the femoral arteries. In many cases the marked spiral form of the vessels is very striking, and it is, as we have said, a direct result of the loss of elasticity of their walls and of the increased blood pressure. The spiral form is most frequently observed in the temporal, brachial, and radial arteries. The examination of the brachial artery is of the most practical importance. Its tortuosity and abnormally strong and visible pulsation is to be accepted as a certain sign of arteriosclerosis. The high-tension hard radial pulse resulting from hypertrophy and hypertonicity of the muscular coat in chronic nephritis must not be confused with real arteriosclerosis. I would like to emphasize the fact that with striking frequency I have found atheromatous changes in the brachial and radial arteries in laborers more strongly developed on the right side than on the left. It appears to me not improbable that this condition has some relation to the greater muscular activity of the right arm.

Although we can often directly demonstrate atheroma in the vessels mentioned, we must always be cautious in deciding from this that there is also an atheroma of the internal arteries, for the radial arteries often feel very rigid, while the autopsy later on shows little or absolutely no atheroma of the internal arteries. In other cases, however, we find at the autopsy marked atheromatous changes in the arteries of the brain, the kidneys, the heart, etc., although the external arteries during life felt perfectly normal. We see from this how hard it is to make an absolute diagnosis of general arteriosclerosis.

It is impossible to give a uniform picture of arteriosclerosis, since its results appear now chiefly in this organ and now chiefly in that, whereby entirely distinct types of disease arise. Hence we must confine ourselves here only to mentioning briefly the most important sequelæ. For the most part, they are described separately in other portions of this work.

In the heart we find a hypertrophy of the left ventricle as a result of the increased resistance to the arterial circulation. This is often apparent during life from the strength of the apex beat and its displacement to the left, and also from the extension of the area of cardiac dullness to the left. On auscul-

tation, the increased tension in the aortic system is made manifest by the strength of the aortic second sound. The examination of the heart, however, is often rendered difficult by the presence of pulmonary emphysema. On the other hand, we sometimes cannot decide how far a manifest hypertrophy of the left ventricle is due to an arteriosclerosis, and not to other coexisting processes, such as contracted kidney. We often find other anatomical changes in the heart besides hypertrophy of the left ventricle. We have already spoken of the important and interesting results of atheroma of the coronary arteries, the formation of the so-called indurations of myocarditis in the heart (see page 414 *et seq.*). Sometimes, from an invasion of the aortic valves by the atheromatous process, we get an insufficiency, or much more rarely a stenosis of the aortic orifice. Of the greatest clinical significance is the sclerosis of the aorta, which generally leads to a diffuse dilatation of the aortic tube, and not infrequently, also, to circumscribed aneurismal pouching. These conditions and their clinical sequelæ will be separately considered in the next chapter.

We have already described the character of the peripheral arteries. The radial pulse is hard and tense, and the wave is either quite large, or, when the tube is narrow, small. Since the wall of the vessel contracts slowly, in consequence of its loss of elasticity, the radial pulse is usually sluggish—*pulsus tardus*. This condition is also pronounced in the sphygmographic tracing, which shows a slow ascent, and a still slower descent, of the pulse curve, and an absence of the elevation in the descending limb of the curve, due to the normal elasticity. The frequency of the pulse is quite different in different cases. Usually there exists a continuous increased frequency of the pulse, between 100 to 120 beats per minute. However, a permanent slowing of the pulse may occur, especially, in all probability, in sclerosis of the coronary arteries. The pulse is very often irregular as a consequence of changes in the heart. We sometimes find an abnormal delay in the radial pulse, or in the pulse in other arteries, in comparison with the heart beat, from the lessened rapidity of transmission of the pulse wave.

Besides the heart, the brain is the chief place in which we observe definite results of arterial sclerosis. The increased tendency to rupture which the atheromatous vessel walls show, and the coexisting heightened blood pressure, explain the comparatively frequent occurrence of cerebral hemorrhages. Cerebral hemorrhages often result from little miliary aneurisms which have formed in the atheromatous cerebral arteries. Atheroma is also the most frequent cause for the formation of foci of softening in the brain, since the arterial changes may give rise to a closure of the cerebral arteries from thrombosis or embolism. Finally, so-called senile dementia, or, at any rate, many cases of that condition, are referable to cerebral atrophy, resulting from the faulty nutrition of the brain through the sclerosed arteries (*cf.* chapter on the Arteriosclerotic Diseases of the Brain).

In the kidneys, too, atrophic processes often develop from the diminution of the blood supply owing to the narrowed lumina of the vessels, and they lead to a special form of contracted kidney. The origin of the granulated "senile kidney" is in large part atheroma of the renal arteries.

The lower extremities display symptoms of arteriosclerosis less often than the parts already mentioned, but if present the symptoms may be very strik-

ing. Here belongs a peculiar group of symptoms, which has been specially studied by Charcot, Erb, and others. It is termed intermittent claudication or lameness, or *dysbasia intermittens angiosclerotica* (Erb). It is sometimes associated with lesions of one or both femoral or iliac arteries, more often with obliterative arteritis of the distal arteries of the lower leg and foot. It usually begins with disagreeable paræsthesiæ of crawling, tickling, or coldness in the feet and calves. If the patient walks even for a short time the symptoms increase, the feet grow cold, pale, and blue, there is actual pain, and the patient becomes completely incapable of walking farther. After a short pause he can resume his walk, but soon he has to stop again. Upon examination we find the feet and legs cold and bluish-red. If we palpate the arteria dorsalis pedis, or the tibialis postica, we can detect no pulsation whatever. This is the most important sign. The course of the trouble is chronic, but decided improvement may ensue under correct treatment (*vide infra*). From a prognostic point of view there is an unfavorable tendency to the development of gangrene of the toes, as the arteritis increases. With regard to causation, an important part in the development of intermittent lameness is played by syphilis, excess in tobacco, glycosuria, damage from thermic influences, and other causes. Conditions analogous to those developing in arteriosclerosis of the femoral arteries occasionally occur in the arms if marked sclerotic disease of the brachial vessels exists.

Fig. 66.—*a*, Arteriosclerosis of the ulnar artery. (Breslau Medical Clinic.)

Sometimes gangrene of the lower extremities occurs as a result of marked atheroma without any previous appearance of intermittent lameness. So-called "spontaneous gangrene," senile gangrene, and diabetic gangrene are probably, without exception, due to primary lesions from arteritis and consequent impairment in the arterial blood supply.

All this shows how different the symptoms of arteriosclerosis may be in different cases. The symptoms in the vascular apparatus often predominate over all others. The heart, which is simply hypertrophied, or which has undergone in part cicatricial degeneration, is finally exhausted, and then all the symptoms of a chronic heart disease develop—dyspnœa, œdema, etc. If there is also albuminuria, a type of disease is produced which resembles that of contracted kidney. In other cases, however, the symptoms in the brain are especially manifest, either alone or in combination with the other symptoms mentioned, and, finally, in rare cases we have the above-described symptoms relating to the lower extremities.

It is worth mentioning that not only the sclerotic changes in the aorta,

but also often the arteriosclerosis of the peripheral vessels, appears distinctly in the radiogram. It is obvious that this fact is of great diagnostic significance. The accompanying Fig. 66 shows an X-ray picture of an atheromatous ulnar artery.

We must remark, in conclusion, that all the results of arteriosclerosis mentioned may be absent for a long time or altogether. Many persons have practically no symptoms at all from their arteriosclerosis, and reach an advanced age, but we must always consider the possibility of the sudden occurrence of severe symptoms, and make our prognosis accordingly.

Treatment.—If we recall the above-mentioned causes of arterial sclerosis, we see at once the possibility and importance of prophylaxis, and of course if the disease already exists we must endeavor to avert these injurious influences, so as to limit the progress of the disease as far as possible. There is little direct influence upon the arteriosclerotic process to be expected from drugs. Iodid of potassium has won the highest reputation in this regard, and it is employed extensively in all cases of arteriosclerosis. Probably it is most efficient in those cases which are associated with a syphilitic taint. It is best to give for some time daily small doses (7.5 to 15 gr. = 0.5 to 1.0 gm.) of sodium iodid or sajodin. The value of the so-called "antisclerosin," a mixture of certain inorganic salts, is quite doubtful. The consideration of many therapeutic details will be found in the respective chapters on the separate localizations of arteriosclerosis. In the symptom-complex known as intermittent lameness, Erb recommends iodid of potassium, or iodid of sodium; local applications of warmth (for instance, by means of the warm pack); galvanic foot baths; such cardiac tonics as strophanthus; and careful regulation of bodily exercise. If gangrene has set in, prompt amputation of the necrotic part is necessary, for we have no means of influencing the process itself.

CHAPTER II

ANEURISM OF THE THORACIC AORTA

(Sclerosis of the Thoracic Aorta)

Ætiology and Pathological Anatomy.—The circumscribed dilatation of an artery is termed an aneurism. The cause of its formation is always to be sought in a primary disease of the vessel wall, which weakens its resistance to the blood pressure. As we have already said in the previous chapter, it is chiefly arteriosclerosis which lies at the foundation of aneurisms in most cases. The same factors, therefore, which favor the origin of arteriosclerosis belong to the ætiology of aneurism (see page 454). This explains why the root of the aorta, where arteriosclerosis is most marked, is also the most common seat of aneurism, and further, why aneurisms are especially seen in advanced life and in men.

Besides arteriosclerosis, syphilis is undoubtedly one of the most frequent causes of aneurism. In my experience, aneurisms developing in early life are almost without exception referable to a previous syphilis. Finally, severe traumata of the thorax must also be regarded as ætiological factors. A slight

rupture of the vessel wall may take place, and at this point the aneurismal dilatation gradually develops. Traumatic aneurisms, to be sure, are quite rare.

The size of aneurisms of the aorta varies very much, of course, in different cases. They most frequently are about the size of an apple or the fist; but in rare cases much larger aneurisms are observed. According to their shape we distinguish the more diffuse or spindle-shaped dilatations from the saccular aneurisms (*aneurisma diffusum seu cylindricum, aneurisma fusiforme, et aneurisma sacciforme*). Intermediate forms and combinations of the different forms occur in manifold ways. There is no sharp line of difference between the spindle-shaped aneurisms and the diffuse dilatations of the thoracic aorta. Thus, we may speak of the clinical picture of "sclerosis of the thoracic aorta," whereby it is only a secondary consideration whether the aorta is at the same time markedly dilated or not.

As we should expect from its origin, we never find the wall of the aneurism formed of normal vascular tissue. The intima almost always shows the same changes that are characteristic of arteriosclerosis, only in a much higher degree. The media, too, is usually changed, and its muscular structure often shows fatty degeneration. The adventitia is usually thickened by chronic inflammatory processes. The media, and sometimes the intima, are in many cases so much atrophied that the wall of the aneurism, at least in part, is formed only of the adventitia.

In the cavity of the aneurism the blood is only partly fluid. We usually find it more or less full of new and old masses of thrombi. The oldest thrombi, which lie upon the wall of the aneurism, are firm, yellowish, adherent to the wall, and sometimes calcified. At other points the thrombi are softened and broken down. The most marked coagulation is usually found in the saccular aneurisms with a narrow entrance, because in this form of aneurism the blood is almost completely stagnant in the aneurismal sac.

Aneurisms of the aorta usually have their seat in the ascending aorta, or in the arch. Aneurisms of the descending thoracic and of the abdominal aorta are far more rare. The following description refers principally to aneurisms at the beginning of the aorta.

We do not give separate consideration to aneurism of the ascending aorta and of the arch of the aorta, respectively, for it is impossible clinically to draw a sharp distinction between them. Other aneurisms will be considered farther on.

Clinical Symptoms.—1. *Subjective Symptoms.*—The subjective symptoms indicative of an aneurism or a sclerosis of the thoracic aorta are sometimes very indefinite, although they often enough point with sufficient probability to the true nature of the disease. Most important are the painful sensations in the front of the chest behind the sternum associated with a certain feeling of distress and oppression. The pains occur especially after exertion. After he has made a few rapid steps, the patient must stand still on the street. Sometimes the pains radiate to the back, or to the shoulders and arms, especially to the left arm. Occasionally, as a result of pressure of the aneurism upon a nerve (intercostal nerves, brachial plexus), the pains assume a true neuralgic character. If there are also associated with these pains other suspicious symptoms, such as palpitation, dyspnœa, cough, dizziness, headache,

general malaise, dyspepsia, etc., a careful consideration of the case and an exhaustive objective examination are urgently demanded. Concerning the occurrence of true anginose attacks in sclerosis of the aorta and the coronary arteries, see page 418.

2. *Physical Signs of Aneurism.*—We begin the objective examination with inspection and palpation. We look for an abnormal pulsation. This cannot be observed in many of the more deeply seated and in the more spindle-shaped aneurisms. In other cases, however, the immediately noticeable abnormal pulsation, to a certain extent, permits of the diagnosis of aneurism at the first glance.

Usually the pulsation appears in the second intercostal space, on the right side, in aneurism of the ascending aorta, and, on the left side, in that of the arch. In aneurisms of the arch, occasionally a pulsation may be felt in the suprasternal fossa upon deep pressure with the finger. If no pulsation is felt with the patient in the dorsal position, he must then be also examined standing, and with the trunk bent forward. Sometimes a distinctly palpable thrill is associated with the pulsation.

In the rare aneurism of the descending thoracic aorta the pulsating swelling may make its appearance in the back, between the vertebral column and the left scapula. If the aneurism has reached a certain size, the pulsating part protrudes as a tumor. The protrusion is either slight, or in many cases it forms a large prominent swelling. This tumor then shows usually a marked pulsation, not only from below upward, but also in a lateral direction, which is of diagnostic significance. In large aneurisms, however, the pulsation sometimes is very weak, and scarcely perceptible, from the formation of many coagula.

The marked prominence of large aneurisms is possible only because the covering parts, not only the muscles and skin, but also the cartilages and bones, the ribs and sternum, are brought to a gradual atrophy and wasting by the persistent pressure. The skin over large aneurisms gradually becomes thinner and thinner, until finally it may even become necrotic.

There is of necessity more or less dullness on percussion over every aneurism of any size, provided it approaches the thoracic wall. The dullness is usually evident in the upper right intercostal spaces, or the adjacent parts of the sternum. Frequently it even precedes the palpable pulsation, although then its significance is usually still very uncertain. Aneurisms of the arch occasionally produce dullness in the first and second left intercostal spaces. To determine the areas of dullness, the entire upper anterior chest wall should be gone over from right to left, the percussion being performed upon the finger held vertically. Small areas of tympanitic resonance should be looked for. I have repeatedly found these in aneurism in the neighborhood of an area of dullness, although they may be present even without the latter. They are undoubtedly due to the retraction of the part of the lung adjacent to the aneurism. In diffuse dilatation of the sclerotic aorta, the presence of sternal dullness can often be demonstrated by careful percussion.

Auscultation gives varying results. In some cases (probably chiefly when many coagula form) we hear nothing at all over the aneurism. In other cases we hear one or two sounds, which are usually the audible heart sounds transmitted. Perhaps a systolic sound may also arise from vibration of the

wall of the aneurism. In other cases we hear a murmur over the aneurism. A dull and usually not very loud systolic murmur often arises from the formation of eddies in the aneurismal sac. If we also hear a diastolic murmur, it is almost always due to a coexisting insufficiency of the semilunar valves of the aorta (*vide infra*).

In those rather frequent cases in which the physical examination leaves us in the lurch, the radiographic examination of the chest furnishes an unusually reliable means of gaining definite knowledge of the condition of the aorta. It can be said without exaggeration that we did not have an idea of the frequency of aneurism until the introduction of radiography. The diffuse and spindle-shaped dilatations of the aorta especially can often be recognized by this means alone; but also numerous sacculated aneurisms, that give no other objective signs, can be diagnosed with absolute certainty by the X-ray examination. The dilatation is recognized in the aortic shadow, and the pulsation can be seen with the fluoroscope.

Fig. 67.—Radiogram of a case of aneurism of the arch of the aorta. (Breslau Medical Clinic.)

An aneurism of the aorta by itself probably never causes such an increased resistance to the blood current as to give rise to the development of a hypertrophy of the left ventricle. Therefore, in many cases of aneurism, particularly of the arch, we find the heart unchanged.

In the quite frequent cases in which hypertrophy of the left side of the heart exists, it may almost always be referred to a coexisting insufficiency of the aortic valves, and sometimes to general atheroma of the arteries. During life a hypertrophy of the heart may be simulated, because the heart is pushed to the left by the aneurism.

In many cases the signs in the peripheral arteries are important. Marked inequality of the pulse in symmetrical arteries is often an especially valuable diagnostic sign. Either the trunk of an efferent vessel is compressed by the aneurism, or the lumen of the exit of the vessel is itself involved in the aneurism, and hence the opening of the vessel is distorted or contracted, or partly stopped by a coagulum. This readily explains why, in aneurism of the ascending aorta, the radial, and sometimes the carotid pulse, are plainly weaker on the right than on the left, as a result of implication of the trunk of the innominate, while in aneurism of the arch or of the beginning of the descending aorta the opposite condition may obtain. Abnormal differences, too, in the intensity of the pulse in the upper and lower halves of the body may arise under some circumstances.

A marked delay of the pulse in the arteries arising below the aneurism is a symptom that is occasionally seen. Thus, we see in aneurism of the arch of the aorta that the left radial pulse is later than the right, and that in aneurism of the descending aorta the pulse in the lower extremities is later than the radial pulse.

3. *Signs of Compression.*—It is quite in accordance with the anatomical relations of an aneurism that numerous neighboring structures are exposed to a greater or lesser degree of pressure from the sac. From this a further train of symptoms may develop. First of all, the adjacent veins (the larger venous trunks in the thorax, the superior vena cava, or one of the innominate veins) may be compressed. The veins swell in the neck, in the upper extremities, or upon the surface of the thorax, according to the seat of the compression. Local œdema may also be produced in this way (œdema of the face, neck, and arms).

The respiratory organs are exposed to the pressure of aortic aneurisms in many ways. Compression of the lungs by large aneurisms actually contributes toward increasing the dyspnœa in many cases. This may be still more distressing if the trachea be compressed. Of the two main bronchi, the left bronchus, which lies beneath the arch of the aorta, is more apt to be compressed. This produces the symptoms of a unilateral bronchial stenosis (*q. v.*). From the constant compression of a bronchus, retention of the secretions, chronic bronchitis, and, finally, atelectasis and fibroid consolidation develop in the corresponding portion of the lung. Thus are explained the peculiar pulmonary complications, such as chronic interstitial pneumonia with chronic bronchitis and pleurisy. These are not infrequent, and we have personally seen a considerable number of examples. Many a case of severe diffuse bronchitis in an elderly arteriosclerotic patient at the X-ray examination (or at autopsy!) turns out to be an aneurism of the aorta. If an aneurism of the concave side of the arch of the aorta transmits its pulsation to the left bronchus lying below, in and thence to the trachea and larynx, a systolic, downward pull of the larynx may be observed—the so-called Oliver-Cordarellis sign [tracheal tug].

The comparatively frequent compression of the left recurrent laryngeal nerve, producing a paralysis of one vocal cord, is of great diagnostic importance. Such a unilateral recurrent paralysis and the resulting discordant, hoarse voice are very often the first symptoms to arouse suspicion of the existence of an aortic aneurism. Paralysis of the right recurrent nerve occurs in aneurisms of the innominate artery.

Pressure on the vagus may occasion changes in the rate of the pulse, and perhaps sometimes attacks of dyspnœa. The dyspnœa in most cases, however, takes the form either of angina pectoris or of cardiac asthma, and is referable to the other coincident lesions of the heart and the coronary arteries.

A very characteristic clinical picture presents itself if the aneurism compresses the superior vena cava. We see, then, all the sequelæ of a venous congestion of the upper part of the body, extreme cyanosis of the neck and face, marked prominence of the veins of the neck, and œdema of the face and arms. As a result of compression of the intercostal nerves or branches of the brachial plexus, most severe and distressing neuralgias arise in the nerve territories affected, and sometimes we see motor paresis in the arm.

Finally, disturbances of deglutition are seen in many cases from compression of the œsophagus. If this be falsely interpreted, it may lead to a mischievous use of the œsophageal sound, as perforation of the aneurism may be caused. Hence, we must always remember this possibility in practice.

Course and Termination of the Disease.—Aneurisms may remain latent for a long time without causing any symptoms. In such cases a sudden perforation may lead to speedy and unexpected death.

In the cases which have shown the above symptoms to a greater or less extent for a long time, and often for years, sudden death quite frequently results from rupture of the aneurismal sac and perforation into a neighboring organ. In perforation into the pericardium death follows almost instantly from cessation of the heart's action. In perforation into the œsophagus a fatal hemorrhage occurs. In perforation of the aneurism into the air-passages (the trachea or bronchi) or into one pleural cavity, two factors, hemorrhage and suffocation, unite in causing death. Often several smaller hemorrhages, which are frequently not noticed or incorrectly interpreted, precede the fatal hemorrhage.

In aneurisms which gradually erode the anterior wall of the chest, the perforation is in rare cases external; but here a sudden, immediately fatal hemorrhage seldom ensues; much more commonly a slowly increasing anæmia develops as a result of repeated slight hemorrhages which may sometimes go on for weeks. Death then is due to the gradually increasing weakness, or to a final severe hemorrhage. Perforation of an aneurism into the right side of the heart, into the pulmonary arteries, or the vena cava, is a rare termination. Here death does not follow at once, but severe general disturbances of the circulation, such as dropsy, soon arise. In many of these rare cases peculiar physical signs also appear—a venous pulse, a loud systolic murmur over the point of perforation, etc.

If, in patients with aneurism of the aorta, death does not ensue from a sudden perforation, the general type of the disease takes a form similar to chronic heart disease. The aneurism, as we have said, is often also combined with aortic insufficiency, or with sclerosis of the coronary arteries and subsequent interstitial myocarditis. The left ventricle gradually becomes paralyzed, and the well-known disturbances of compensation set in—increasing dyspnœa, œdema, etc. In other cases the patient gradually becomes duller and weaker from the distressing pain, the sleeplessness, and the other symptoms, and dies with the signs of increasing general weakness.

Recovery from aneurism of the aorta scarcely ever occurs.

Diagnosis.—The diagnosis of aneurism of the aorta can in many cases be made with great ease and certainty, but in other cases it is very difficult and even impossible. If the direct physical signs are plain, especially if we feel an abnormal pulsation, we shall not be apt to commit an error; but the diagnosis presents great difficulties in those cases in which the aneurism is not accessible at all, or accessible only with great difficulty, where it merely causes indefinite symptoms, pain in the chest, occasional oppression, symptoms of pressure on neighboring organs, etc. A very stubborn intercostal neuralgia, which no remedy can relieve, may for a long time be the only symptom, often misinterpreted, of a latent aneurism. The disease is often not recognized, however, because in such and similar cases we do not generally

think of the possibility of an aneurism, and hence we neglect a careful examination of the heart and the arteries, and also the search for other symptoms of compression, such as paralysis of the vocal cords, and above all the most significant X-ray examination.

The distinction between aneurism and other tumors in and about the thorax sometimes presents difficulties in diagnosis. Mediastinal sarcomata and abscesses, circumscribed empyemas, tumors arising from the sternum, or new growths in the lungs and bronchial glands, may all give rise to confusion. We can scarcely lay down any general rules for diagnosis, since the conditions differ in almost every case. If we feel a swelling, its pulsation is the symptom which points most to an aneurism, but we must be certain that the pulsation is not merely transmitted, but that it really takes place in all directions within the swelling itself [expansile pulsation]. We must also consider the auscultatory symptoms, the condition of the heart and the arteries, and any symptoms of compression, and above all the result of radiography.

Treatment.—Many attempts have been made to bring about an obliteration of the aneurism, and thus a recovery. Although the methods of treatment aiming at this have obtained decisive results in the aneurisms of peripheral arteries, their results in aneurism of the aorta are still of a very doubtful character; yet we are always justified in any given case in trying one of the methods recommended.

Persistent compression by a pad can, of course, be employed only in those cases where the aneurism projects at one part of the chest wall. The pressure, however, usually causes great pain, and hence is ill borne.

Tying the carotid, the subclavian, or both vessels, has also been repeatedly performed in aneurism of the arch of the aorta, sometimes with apparent success, but oftener without any benefit.

"Acupuncture" of the aneurism (Velpeau) consists in inserting a needle or an iron wire into the aneurismal sac in order to excite coagulation in it. The results obtained by it in aneurism of the aorta are not very encouraging.

Better results are reported from galvano-puncture. Two needles inserted into the aneurism are connected with the poles of a galvanic battery, by which a weak current is passed through the aneurism. Here we must regard the chemical and electrolytic action of the current as well as the mechanical action of the needles. At present, however, the method is scarcely used.

Injections of chemical substances into the aneurismal sac, in order to produce coagulation, are dangerous, since the coagula caused by them may give rise to emboli. Hence we have abandoned making trial of liquor ferri sesquichloridi and similar substances. We can better recommend injections of ergotin into the vicinity of the sac, 2 to 5 gr. (gm. 0.1 to 0.3) of the aqueous extract of ergot dissolved in water or glycerin, injected every day or two. This method was first employed with success by Langenbeck in peripheral aneurisms.

Most used at present is the injection of gelatin, first recommended in France. Every five to eight days 6.5 oz. (gm. 200) of a one- to two-per-cent gelatin solution is injected subcutaneously into the breast, but not in the immediate vicinity of the aneurism. Coagulation in the aneurism sac is

supposed to be stimulated by the gelatin. We have obtained some apparently good results by this method, though we have also had numerous failures.

We can expect little action on an aneurism from the use of internal remedies. Especially recommended is the long-continued use of iodid of potassium, iodid of sodium, sajodin, etc., which have obtained a great reputation in all arteriosclerotic lesions and, in particular, in syphilitic disease of the arteries (*vide supra,* ætiology of aneurism). Yet it is easy to see why one should not expect too much from the iodids, although at times some amelioration of the symptoms can be obtained by a systematic iodid treatment. The pains may cease and the feeling of distress may be alleviated.

The treatment of aneurism, therefore, will be mainly symptomatic and general. The main thing, as soon as the diagnosis is certain, is to protect the patient as much as possible from all injurious influences, especially bodily exertion. Cold applications, locally (ice bag, Leiter coil), sometimes act very favorably. If the tumor protrudes, a protective pad of lead, celluloid, or the like, may be applied. The pains, anginal attacks, and dyspnœa are treated on general principles (morphin, nitroglycerin, digitalis, etc.). Treatment is powerless against internal perforations.

[Tufnell's method, so-called, which has given good results in abdominal and peripheral aneurisms, proves sometimes useful in palliating the symptoms and lengthening the course of aortic aneurism. The aim of this method is to diminish the force and rapidity of the circulation, and, if possible, to increase the fibrinous deposit. It is carried out by enforcing absolute rest in the recumbent position, and by limiting the amount of food, especially of liquids. About ten ounces of solid food and eight of liquid are allowed daily, divided into three meals.]

CHAPTER III

ANEURISMS OF THE OTHER VESSELS

Aneurism of the Abdominal Aorta.—Its favorite seat is the vicinity of the cœliac axis. In many cases it may be felt through the abdominal wall as a pulsating tumor, over which a systolic sound or a whirring murmur can be heard. The possible symptoms of compression are very numerous. The stomach, intestine, and liver (jaundice) may be implicated. Pressure of the aneurism upon the nerve trunks, or even pressure on the spinal cord after gradual erosion of the vertebræ, with consequent severe neuralgia, paralysis, etc., has been repeatedly observed. Death usually ensues from rupture of the aneurismal sac and internal hemorrhage.

Aneurism of the trunk of the innominate is rare. Its symptoms are very much like those of an aneurism of the arch of the aorta. If we feel a pulsating tumor, it is usually situated somewhat higher up than the aneurism of the aorta, in the first right intercostal space, or the tumor even extends into the supraclavicular fossa. In rare cases aneurisms of the subclavian and of the carotid have been observed. We have ourselves seen an aneurism of the internal carotid the size of a cherry pressing on the Gasserian ganglion, which caused an extremely severe trigeminal neuralgia lasting for years.

Aneurism of the pulmonary artery may appear as a pulsating tumor in the second left intercostal space. It is usually impossible to distinguish it with certainty from an aneurism of the aorta.

We have already mentioned, in the description of pulmonary tuberculosis, the great importance of small aneurisms of the branches of the pulmonary artery in pulmonary cavities as a frequent cause of hemorrhage.

Aneurisms of the arteries of the brain, which are relatively most frequent in the basilar artery and the artery of the fissure of Sylvius (the middle cerebral artery), may cause severe cerebral and bulbar symptoms (see Vol. II). As has already been mentioned, miliary aneurisms of the cerebral arteries play an important part in the ætiology of cerebral hemorrhage (*q. v.*).

The symptomatology and treatment of aneurisms of the peripheral arteries belong to the domain of surgery.

CHAPTER IV

RUPTURE OF THE AORTA

A RUPTURE of a previously healthy aorta, with fatal hemorrhage, after violent traumatic influences, has been seen only in a very few cases. In the majority of the very rare cases of rupture of the aorta we have to do with a vessel that is already atheromatous. In some cases a special exciting cause is present, and in others it is absent. We once saw sudden death caused by rupture of the ascending aorta in a young man about twenty-five, who before that seemed perfectly healthy. No trace of atheroma was found; but at the point of rupture there was a slight protrusion and a decided thinning of the wall, which was probably congenital. The formation of a so-called dissecting aneurism, which has often been seen in the aorta, is of anatomical interest. Here only the intima and media are torn. The blood burrows between them and the adventitia or between the layers of the media. Most of the cases of dissecting aneurism of the aorta also result, like rupture of the aorta, in sudden death. In many cases death results from the secondary perforation of the aneurism into the pericardium. On the other hand, a sort of recovery from dissecting aneurism may occur. A secondary perforation takes place into another part of the aorta itself (Boström). Cases of this sort were formerly more than once mistaken for double aorta. If the capsule containing the blood is preserved for a considerable length of time, the symptoms may assume the character presented in ordinary aortic aneurism.

CHAPTER V

NARROWING OF THE AORTA

CONGENITAL narrowness of the aorta and its branches is a condition to which Rokitansky first, and later Virchow, have directed attention. We find this anomaly especially in those, mostly women, who during life have shown

the signs of persistent chlorosis. Sometimes such persons are backward in their whole development; they retain a puerile habit, and show a defective development of the genitals. They often suffer from palpitation, faintness, and a tendency to hemorrhages. In many cases the heart is also small, but in others it is dilated and hypertrophied. We have already pointed out (page 428) that congenital hypoplasia of the aorta may be regarded as the cause of certain cases of "idiopathic hypertrophy of the heart." Valvular disease of the heart has been repeatedly found combined with general narrowness of the arterial system. During life this anomaly of the vascular system may sometimes be suspected, but it can never be recognized with certainty.

Narrowing of the aorta at the point of insertion of the ductus arteriosus is a lesion observed in rare cases, whose origin probably always falls in the period directly after birth, and is associated with obliteration of the fetal ductus arteriosus. Other congenital anomalies of the heart are usually present at the same time. If the narrowing of the aorta is not very marked, it may be properly compensated by a secondary hypertrophy of the left ventricle and the development of a collateral circulation. The latter is brought about by dilatation of the anastomoses between the first intercostal artery, the dorsalis scapulæ, the subscapularis, and the transversalis colli on one side, and the lower intercostal arteries, which come off from the descending aorta below the narrowing, on the other. Anastomoses are also formed between the mammary and the superior epigastric on one side and the lumbar and femoral arteries on the other. During life the dilated arteries are prominent, in part abnormally tortuous, and perceptibly pulsating, especially the dorsales scapulæ, the subscapulars, the mammaries, and the epigastrics. In some cases a systolic murmur has been heard over some of these vessels. The pulse in the arteries of the lower extremities, the femoral and popliteal, is very weak and scarcely perceptible.

In many cases the collateral circulation is so complete that the person affected may feel no subjective disturbance at all, and may attain an advanced age, but in other cases disturbances of the circulation appear sooner or later, and the patient finally succumbs to dropsy. Sudden death from rupture of the heart or of the aorta has also been observed.

IV. DISEASES OF THE DIGESTIVE ORGANS

SECTION I

Diseases of the Mouth, Tongue, and Salivary Glands

CHAPTER I

CATARRHAL STOMATITIS

(Catarrhal Inflammation of the Mucous Membrane of the Mouth)

Ætiology.—Inflammation of the buccal mucous membrane is not infrequently the direct result of mechanical or chemical causes. As mechanical causes we may mention particularly the sharp edges of broken or carious teeth. It should be said that often infectious organisms (*vide infra*) are active at the same time, because they gain a much easier foothold on account of the presence of bad teeth. Chemical irritation may come from highly spiced food, or from tobacco-chewing or excessive smoking. Chemical and mechanical irritation provokes stomatitis in many trades which expose the artisan to lime, copper, coal, or other varieties of dust. In drunkards we find not infrequently a chronic stomatitis, which is characterized by a thickly coated, moist, and shiny tongue, which is somewhat swollen and exhibits indentations on its side due to its pressure against the teeth. Inflammation of the mucous membrane of the mouth from poisoning with corrosive acids or alkalies is also the result of direct chemical irritation. It may take the form of severe ulcerative stomatitis (see the following chapter). The mercurial stomatitis, which results from acute or chronic poisoning with mercury, is caused indirectly by the particles of mercury deposited from the blood in the mucous membrane. This mercurial stomatitis when severe always takes the form of ulceration or necrosis. The stomatitis attendant upon the cutting of teeth in children will be discussed below.

In many instances stomatitis comes from a direct propagation of inflammation from neighboring parts. It thus forms a frequent complication of pharyngeal catarrh, and less often of rhinitis.

Infection plays an important part in the ætiology of stomatitis. The local inflammation may be merely part of a constitutional infectious disease, as in measles, variola, and syphilis. Stomatitis is still more frequently a complication of some severe and protracted illness, when the mouth is not properly attended to and cleansed. The bits of food and the mucus quickly begin to decay. Great numbers of fungi and bacteria invade the buccal cavity, and

excite inflammation in its mucous membrane. Even the cases of primary stomatitis which sometimes occur are probably all of an infectious character. They usually soon assume the ulcerative form.

Clinical History.—The usual symptoms of an inflammation of mucous membrane—namely, redness, swelling, and increased secretion—are exhibited in stomatitis. The redness is usually most intense on the inside of the cheeks and on the gums. Indeed, we have the special name—gingivitis—for inflammation of the latter. The swelling is best shown by the indentations made by the teeth in the cheeks and the edges of the tongue. The tongue and gums are smeared with mucus. There is often considerable salivation. If the inflammation is more active, we find a mucopurulent coating on a greater or less portion of the membrane. The tongue is almost always thickly coated. If we scrape off a little of the coating and put it under the microscope, we find a great abundance of pavement epithelium, in part fatty degenerated, pus, micrococci, and remains of food. White spots made up of epithelium may also be seen on various parts of the oral mucous membrane. Here and there little vesicles appear which burst and leave superficial ulcers.

The local discomfort of severe stomatitis is by no means trifling. There is burning pain, which interferes with taking food, and usually the processes of decomposition occasion a constant bitter or disgusting taste in the mouth, as well as a foul and offensive breath.

The duration of the disease depends on the nature of the immediate cause or the character of the primary disorder. Usually a stomatitis which gets well in one or two weeks is called acute, and a more tedious attack, chronic. The chronic form is seen in topers, inveterate smokers, and persons with bad teeth. It may last for years, with the symptoms described above, only milder. (For lingual psoriasis, *vide infra*).

Treatment.—If the inflammation is considerable, the diet must be liquid. Sometimes cold drinks are most agreeable, but usually lukewarm are preferred. Often the pain is relieved by taking from time to time a sip of iced water or a bit of ice; but in most cases the patient will prefer to rinse the mouth with lukewarm water. The local use of cocain acts beneficially in severe pain. The important indication, to keep the mouth as clean and pure as possible, is best met by having the mouth frequently rinsed out with a two-per-cent solution of chlorate of potash, or one or two teaspoonfuls of a one-per-cent solution of permanganate of potash in a glass of water. It is also advisable to rinse the mouth with a two-per-cent solution of peroxid of hydrogen (preferably chemically pure hydrogen peroxid (Merck), dispensed in thirty-per-cent solution), and with thymol, which latter is the chief constituent of Miller's mouth wash, the ingredients of which are: Thymol, 0.25 part; benzoic acid, 30 parts; tincture of eucalyptus, 4 parts; distilled water, 750 parts. In children who cannot do this, the mouth is to be carefully washed or sprayed. If the gums are spongy, they should be painted with a mixture containing equal parts of tincture of myrrh and tincture of rhatany. If there are superficial ulcers scattered about, it is sometimes an excellent plan to pencil them with a ten-per-cent borax-glycerin solution or to touch them lightly with lunar caustic, to hasten their healing.

Chronic stomatitis is often very obstinate, resisting all sorts of treatment for a long time. The first thing is to remove any such injurious agencies as

tobacco or bad teeth. It is recommended, besides the above-mentioned remedies, to swab out the mouth with a solution of corrosive sublimate (1 to 5,000) or of lunar caustic (1 to 30 to 50).

CHAPTER II

ULCERATIVE STOMATITIS

(Stomacace)

Ætiology.—By ulcerative stomatitis is meant a severe disease of the buccal mucous membrane, with superficial necrosis and the consequent formation of ulcers. The abnormal processes are not in all cases identical, and their course may vary. Still, it is probable that infection is the important factor, at least in the primary cases. The disease has repeatedly been epidemic, chiefly among soldiers in barracks or on a campaign, and among the inmates of jails. We have repeatedly seen sporadic cases of primary ulcerative stomatitis with fever. In children the disease is especially common at the time of the second dentition. Here, too, endemic and contagious influences are frequently in evidence.

Mercurial stomatitis is, from a practical point of view, the most important of those forms of ulcerative stomatitis which are referable to definite chemical action. It is due to the inhalation of the fumes of mercury in those whose trade or scientific occupation exposes them to this danger, and very frequently also to the therapeutic employment of mercury in the form of calomel or mercurial ointments, and the like. (For the scorbutic form, see chapter on Scurvy.)

Symptoms.—The disease usually attacks the gums of the lower jaw first, gradually spreading thence to neighboring portions of the lips and cheeks. Those portions of the mucous membrane of the mouth where the teeth are absent are, strange to say, almost always spared. The tongue and palate are likewise generally not very much affected, though often the seat of a simple catarrhal inflammation. In severe mercurial stomatitis the gums are especially apt to be affected. The ulcers also show a preference for the angles of the lower jaw, and those portions of the mucous membrane of the cheeks which rest upon the teeth.

Inspection shows that the mucous membrane in the places mentioned has a pasty, purulent coating. The gums are swollen, spongy, and red, and bleed easily. The mucous membrane on the edges of the gums becomes necrotic, giving rise to profuse purulent secretion and the formation of ulcers. The teeth are so loosened by gingivitis that they may even fall out. There is usually profuse salivation. The lymph-glands at the angle of the lower jaw and on the chin are generally swollen. The breath is very offensive, poisoning the air of the whole room.

The local discomfort of the patient is the same as in simple stomatitis, only much worse. It is very difficult to take nourishment. In many cases there are marked constitutional symptoms. The patient feels very weak and languid. The primary, and sometimes also the toxic, cases of ulcerative

stomatitis are associated with moderately high fever—100.5° to 102° F. (38° to 39° C.). Now and then severe symptoms of constitutional sepsis have followed the disease.

The course of ulcerative stomatitis is favorable in the great majority of cases. With good treatment and nursing, the ulcers gradually clean up, and at the end of one or two weeks recovery is complete. Exceptionally, the disease may be more chronic. The most frequent way in which recovery is delayed is that the disease extends to the periosteum of the lower jaw, causing necrosis of small portions of the bone, which must be expelled before the patient gets well.

Treatment.—The treatment does not differ essentially from that of the milder forms of stomatitis. The mouth must be still more carefully and more frequently cleansed and disinfected. Good results are obtained from potassium chlorate, permanganate of potash, boric acid, peroxid of hydrogen, and similar remedies. Some authors recommend the simultaneous internal administration of potassium chlorate on account of its subsequent excretion through the saliva, but it is usually useless and not entirely devoid of danger, as it has repeatedly caused poisoning.

As to prophylaxis, we should mention that all patients who are using mercury should, whenever possible, have their teeth first put in good order by a dentist, and should employ a gargle of potassium chlorate faithfully from the beginning of treatment, in order to prevent the occurrence of mercurial stomatitis. If salivation occurs, the mercury must be stopped.

CHAPTER III

APHTHOUS STOMATITIS

(Aphthæ. Disseminated Fibrinous Stomatitis)

Aphthous stomatitis is characterized by roundish spots upon the mucous membrane, grayish white, and of small size, unless made larger by the confluence of several into one another. They usually have a narrow, red areola. They are most numerous on the edges and dorsum of the tongue and on the frænum, but they also occur on the lips and cheeks. The attempt to remove the white spot with forceps never succeeds, but it causes bleeding. In addition to the genuine aphthæ there are almost always the signs of a common stomatitis, which may be mild or severe. The white spots are due in part to a thickening and opacity of the epithelium, and in part are said to be caused by the formation of a fibrinous exudation, which penetrates the most superficial layers of the mucous membrane. If the necrotic epithelium is cast off, the aphthous spot develops into a little ulcer, which often heals promptly, but which may be very obstinate.

The disease occurs chiefly in children, and especially frequently at the time of the first dentition. The child is usually restless, often somewhat feverish, and evidently suffers pain when nursing. Generally there is considerable salivation. The lymph-glands may be a little enlarged. On the outer skin in the neighborhood of the mouth there may also appear a few

blisters and pustules. These, as a rule, have no connection with herpes, although a real mucous-membrane herpes does exist, and now and then genuine herpes may appear in association with aphthous stomatitis. The disease is not rare in adults. Many individuals are especially liable to it, and very frequently have little, white, and often very painful spots here and there on the tongue or elsewhere in the mouth. These have a tendency to develop into ulcers which are usually superficial, but sometimes deep. They may prove very troublesome, from their frequent recurrence and the hindrance they cause to speaking and mastication.

With the exception of the form just described, which is constantly relapsing, aphthæ almost always run a favorable course. There is usually complete recovery in a week or two. The treatment of children consists in carefully washing out the mouth with cold water or mild disinfecting lotions. If the spots do not disappear, we can paint them with a solution of borax (1 to 30), of permanganate of potash (1 to 150), a five-per-cent solution of sulphate of zinc. If some of the places are especially painful, particularly in adults, we may touch them with lunar caustic, when they usually are soon cured.

It is often a very difficult matter to treat chronic, recurrent, aphthous stomatitis, and the ulcerations which are occasioned by it in adults. Cauterization with lunar caustic, chromic acid, and similar agents usually does more harm than good. None of the ordinary mouth washes has any great effect. A much-praised remedy is the chewing of bilberries, either fresh or boiled, for several minutes several times a day, and we have ourselves tested it in some cases with good results. Some physicians also recommend the internal use of iodid of potassium, or small doses of calomel. It is remarkable that habitual aphthous stomatitis sometimes ceases completely for months—for example, upon change of residence or during a visit to the country—but then appears afresh without special cause.

In conclusion, as to ætiology, infection is a most likely cause, if we consider that small epidemics or endemics have repeatedly occurred. Lately attention has been called to the possibility that the milk of cows suffering from hoof-and-mouth disease may be a source of infection. That infection of this sort may occur seems to us indubitable, from the experience of others, as well as our own. In such cases there is not only a severe aphthous stomatitis, but a development of vesicles, pustules, or even extensive purulent inflammation affecting the fingers and nails, and occasionally the hands, arms, and feet, and other parts of the body surface. Frequently, at the same time, slight febrile symptoms and gastro-intestinal disturbances occur. Most cases of this kind, though, pursue a favorable course, but a few epidemics have been described in which the disease became dangerous to life because of constitutional sepsis.

Of course, it is obvious that not all cases of aphthous stomatitis have an ætiological relationship to the hoof-and-mouth disease of animals. In the usual milder forms there are probably other perhaps not always identical infection agents.

There is a special form known as Bednar's aphthæ. In newborn children white patches are not infrequently found lying symmetrically on both halves of the hard palate near the alveolar processes, and persisting till about the third month. These plaques are not syphilitic, although they were formerly in some cases thought to be so. They are probably due merely to the tongue

pressing upon the thin mucous membrane during nursing. Generally they do no harm; but in marantic, neglected children, they may develop into quite deep ulcers. In that case, repeated cauterization with a five-per-cent solution of argentic nitrate is required.

CHAPTER IV

THRUSH

(Soor. Muguet. Sprue)

Ætiology.—Weak and artificially nourished children are particularly liable to this disease; but it also attacks adults who are suffering from phthisis, carcinoma, and severe typhoid or typhus fever. In it grayish-white deposits are developed upon the buccal and pharyngeal mucous membrane. The microscope shows these collections to be fungi (Fig. 68); there are a multitude of oval spores, or conidia, and a tangled mass of long mycelium threads. The botanical position of the thrush fungus (oidium albicans) is not yet entirely clear. Grawitz maintained that the thrush fungus is a sprouting fungus, and nearly related to the mold fungi (mycoderma vini,[1] or saccharomyces albicans—Rees). It seems to be a sort of transitional form between the sprouting fungi and the thread fungi, inasmuch as it appears both in the yeast form and also in the form of thread-like mycelia. Plaut, according to his investigations, makes the thrush fungus identical with the monilia candida, which belong to the Torulaceæ. At any rate, the thrush fungus is widely distributed, for the development of thrush upon the mucous membrane of the mouth and throat is a frequent phenomenon, especially in ill-nourished children. Nursing bottles and the nipples used upon them are probably not infrequently the agents by which the disease is conveyed.

Fig. 68.—Thrush fungus from mouth.

Symptoms.—The mucous membrane of the tongue, cheeks, and soft palate is usually somewhat red and swollen. Upon it we see at first small, punctate, white spots, which may gradually spread. When this coating, which is at first white, becomes more extensive, it assumes a more dirty yellow or brownish color. Microscopic investigations have shown that the fungus develops first in the middle layers of the epithelium. From this starting point it grows not only upward, but also downward into the mucous membrane. If the growth is abundant, it is easy to scrape off the upper layers, and make a

[1] Mycoderma vini is that fungus which is found in the development of vinegar from alcohol, when alcoholic beverages sour.

diagnosis by aid of the microscope. In exaggerated cases the growth may even extend from the pharynx into the upper part of the œsophagus and the entrance of the larynx; but we only rarely find thrush in the larynx itself, or the nostrils, or the stomach and intestines—briefly, in places where there is cylindrical epithelium. In exceptional cases the thrush fungus may extend its growth through the wall of a blood vessel, and so reach the circulation. Usually thrombosis of the affected vessel takes place, but the metastasis of thrush to the brain and the kidneys has been observed. If abscesses are associated with these processes they depend upon the simultaneous intrusion of pyogenic cocci.

As a rule, thrush is accompanied by a more or less severe stomatitis. The fluids of the mouth have an acid reaction. Nursing, or chewing, and swallowing are painful. Still, it is a question whether the stomatitis is due to the fungus, or whether it prepares the territory for the fungus to settle in. Nursing infants, who suffer from thrush, often have diarrhea or marasmus at the same time, which latter affections are more probably the cause than the result of the thrush. If vigorous and healthy sucklings are attacked by thrush, the disease is usually quite harmless, quickly vanishing if proper cleanliness is maintained. In sickly children, particularly if bottle fed, the appearance of the disease is very ominous. The children become very restless and hoarse. The mucous membrane of the mouth and pharynx is dry and of a dark color. The ingestion of food is almost impossible. In adults, as we have said, thrush is with few exceptions confined to persons greatly prostrated by such diseases as severe typhoid fever or tuberculosis, and from this point of view it is an unfavorable symptom.

Treatment.—To prevent the development of thrush in children, the mouth must, if possible, be wiped out, each time they drink, with a cloth wet in cold water; and if adults are very ill, they require equal attention in this regard. As soon as we see the first traces of the disease, we should touch the parts attacked with a brush wet in an aqueous solution of borax (1 to 20). Honey should not be added to the borax solution, as is often unwisely done. Another good solution is bicarbonate of soda (1 to 10), permanganate of potash (1 to 150), or, in severe cases, bichlorid of mercury (1 to 100,000). If the thrush extends into the œsophagus an efficient remedy (Baginsky) is said to be the internal administration of resorcin, in a one-half- to one-per-cent solution, the dose being a teaspoonful every two hours. The food should contain as little sugar as possible, as this furnishes a favorable culture medium for the thrush fungus. Mild cases can usually be cured by the remedies named. If the thrush has once got a vigorous start in the mouth of marantic children, or of adults suffering from an incurable disease, it must be confessed that we often fail to check its growth.

CHAPTER V

DISEASES OF THE TONGUE

Inflammation of the true lingual parenchyma is quite rare, although the tongue's mucous surface is frequently involved in the various diseases of the mouth.

1. **Acute Parenchymatous Glossitis.**—This is the name given to an inflammatory infiltration of the whole or a part of the tongue, usually ending in abscess. A frequent cause is the sting of a bee or wasp, or it may follow burns or severe cauterization. In the rare instances when it is apparently spontaneous, it is probable that some little wound has afforded ingress to the inflammatory poison.

An important rôle seems to be played by the lymph follicles in front of the attachment of the epiglottis—the so-called lingual tonsil, which may serve as a port of entrance for the infectious material.

The symptoms of acute glossitis are very violent in the severer cases. The tongue is enormously swollen, so as sometimes to protrude from the mouth. It has a thick, soft, purulent coating, and sometimes presents excoriations and ulcerations. The patient has violent pain. Talking and eating are almost impossible. There is usually catarrhal inflammation of the rest of the mouth and profuse and very annoying salivation. The tongue may swell so much as to cause dyspnœa and more or less suffocation. The submaxillary lymphatic glands are also more or less swollen. There is usually fever.

Treatment consists in the employment of ice and the various mouth washes (*vide supra*). As soon as fluctuation is obtained, we must give exit to the pus. Often, however, the abscess opens spontaneously.

Frequently the swelling is very distinctly limited to one half of the tongue. Evacuation of the pus is usually followed by a rapid abatement of the discomfort and complete recovery.

2. **Glossitis Dissecans.**—This is a chronic disease, of comparatively rare occurrence and unknown ætiology. It causes the gradual development upon the surface of the tongue of a number of deep fissures and indentations, giving the organ an uneven and ragged look. The pain is due to the frequent presence of excoriations and ulcers in these fissures.

The trouble is not intrinsically dangerous, nor does it need special treatment. We must try the same remedies which have been mentioned above in connection with the various forms of stomatitis, such as antiseptic mouth washes, the chewing of bilberries, etc. If ulcers are present they sometimes require cauterization with lunar caustic.

3. **Lingual Psoriasis.** (*Tylosis; Ichthyosis linguæ et oris.*) **Leucoplacia.**—*Psoriasis linguæ*, again, is a superficial disease, the ætiology of which is unknown. It consists in localized circular hyperplasiæ of the epithelium of the tongue, rarely conjoined with similar spots upon the cheeks and lips. The central portion of mucous membrane which is surrounded by this whitish, slightly raised, and more or less circular, wall of epithelium, is abnormally smooth and of a reddish color. Usually the tongue takes on a resemblance to a map (*lingua geographica*). The disease generally persists for years, and without symptoms except in severe cases. Often it is found by chance in individuals who have known nothing about the unusual appearance of their tongues. Still, it may cause a hypochondriac endless anxiety, especially if he takes it to be syphilitic.

This last statement applies still better to a peculiar disease allied to psoriasis. It is called *leucoplacia*, and affects the mucous membrane of the tongue and mouth. Usually it causes the appearance, on the lateral borders of the tongue, of dull whitish spots, which have the look of scars, and are

generally somewhat notched. As a rule, the lower surface of the tongue and the cheeks display at the same time similar white spots, which are evidently due merely to thickening of the epithelium. Certain spots may disappear, but they are sure to be replaced by others, so that, so far as has yet been observed, the disease must be regarded as incurable. Still, it is not of great importance, as a rule, for in many cases the local discomfort is very slight. If the indentations along the sides of the tongue become cracked or ulcerated, then there may be great pain. Leucoplacia is very often associated with bodily states of neurasthenia, or hypochondriasis, particularly if the malady appears in a person with a syphilitic taint, which happens with remarkable frequency. But it is certainly not to be regarded as a tertiary symptom, rather perhaps as a sort of sequel to syphilis, although many cases have no connection whatever with the latter disease. Antisyphilitic treatment is invariably useless. It is for this very reason important that the physician should be well acquainted with leucoplacia, that he may spare his patient needless anxiety and ineffectual, or perhaps actually harmful, treatment with mercury. The disease has no relation to excessive smoking, one proof of which is that we have seen the condition in women. The only danger of leucoplacia is that sometimes lingual carcinoma may eventually develop on the spot affected with it. We have in this case to deal with a process which is analogous to the development of ventricular carcinoma in the scars of old ulcers, or of cancer of the gall bladder subsequently to gallstones. Treatment is, as we have said, usually unsuccessful. Still, thorough cleanliness and good care of the mouth may avert any great discomfort. We may try the effect of painting the spots with a four-per-cent solution of borax, or a five-per-cent solution of chromic acid. The use of an alcoholic solution of salicylic acid (salicylic acid, 1 part to 5 parts each of spirit of wine and distilled water) is said to be still more efficient. Lately the frequent chewing of boiled bilberries has been recommended for leucoplacia, and we have had good results from this procedure.

4. **The Black-Hair Tongue.** (*Melanotrichia linguæ.*)—Sometimes we find at the base of the tongue a circumscribed area which appears to be covered with short black hairs. Closer examination shows that we have to deal with unusually long and dark pigmented filiform papillæ. The condition is entirely harmless and causes no symptoms or only insignificant ones. It is usually cured by painting with a ten-per-cent alcoholic solution of salicylic acid, or, if need be, by curetting with a sharp spoon. Another variety of black pigmentation of the tongue has been occasionally observed due to a black fungus (*mucor niger*).

CHAPTER VI

NOMA

(Cancrum oris. Water-cancer)

Noma is a gangrene of the cheek, attacking chiefly feeble and sickly children. The disease is rare. It may be primary, but it is usually a sequel

of severe diseases, such as measles, scarlet fever, typhus and typhoid fevers, and pneumonia. Now and then it has been observed in adults. *A priori,* it is extremely probable that noma is due to some parasitic microörganism, although the infectious agents have not been definitely determined. Several investigators have suggested the hypothesis that the exciting cause of noma is identical with the infectious agent of the so-called "calf diphtheria," the *Bacillus necroseos.* It deserves mention that noma is said to occur with much greater relative frequency in moist regions along the coast—for example, in Holland—than in Germany.

The disease begins, without any evident occasion, in an insignificant spot of gangrene on the inner surface of the cheek—that is, in the mucous membrane. It is usually situated near the corner of the mouth. Externally, the parts are soon swollen by collateral œdema, and the whole cheek gradually becomes hard and infiltrated. At first, all we see upon the mucous membrane is a dirty greenish spot not much larger than a silver dime, but soon the whole cheek and the neighboring parts are one mass of gangrene. Bits of dead tissue come away, and foul-smelling ichor flows constantly into the mouth. The collateral œdema may finally pervade the entire half of the face. The neighboring lymph-glands are always greatly swollen.

This condition is, as a rule, accompanied by fever, often reaching or exceeding 104° F. (40° C.). The general health may indeed for a time be astonishingly little affected; but gradually prostration comes on, or even general sepsis develops, with fever, stupor, and delirium. Frequently lobular pneumonia, which may have a gangrenous character, is produced by the inhalation of sloughing bits of tissue; and often the ichor, being swallowed, excites violent and offensive diarrhea. The local discomfort is not really very considerable in most cases, compared to the severity of the disease. There may even be no pain felt whatever.

The prognosis is almost always fatal. Death sometimes occurs suddenly from collapse. Sometimes it comes at the end of three or four weeks, from a gradual sinking of the bodily powers. Recovery has been seen in only a few cases; there is a line of demarcation formed, the sloughs come away, and a slow convalescence follows, leaving extensive and usually very disfiguring scars behind.

Treatment must have for its chief object to check further extension of the gangrene, by removing all parts that are already destroyed. Local cauterization with concentrated hydrochloric acid, or fuming nitric acid, or lunar caustic, or chlorid of iron, is usually futile. It is probably the best way to remove all the gangrenous portion by means of Paquelin's thermocautery. At least in the early stages of noma we may hope something from this method of treatment; but if the case is far advanced, we can hardly expect to accomplish much.

We should also disinfect the mouth as thoroughly as possible. The most efficient means is to syringe it out with solutions of peroxid of hydrogen or permanganate of potash. We should do our best to maintain the patient's strength.

CHAPTER VII

PAROTITIS

(Mumps)

Parotitis, or inflammation of the parotid gland, appears not only as a peculiar, primary, infectious disease, usually epidemic, but also as a secondary complication of numerous other severe diseases. These two forms should be considered separately.

1. IDIOPATHIC, PRIMARY PAROTITIS (EPIDEMIC MUMPS)

Ætiology.—The disease occurs in epidemics that, although not very frequent, may be quite extensive. Endemics also occur, in barracks and schools; and here and there a sporadic case is seen. Children and young adults are most liable to it. Nursing infants enjoy a marked immunity, as well as elderly persons. Males are much oftener attacked than females.

There can be no doubt that mumps is a specific infectious disease, and claims have already been made of the discovery of its specific bacillus, but these lack confirmation. Still, it is natural to suppose that the infectious matter reaches the gland by way of Steno's duct. Numerous observations support the view that the disease is directly contagious; but the degree of contagiousness is not great. Perhaps the saliva of patients has something to do with the extension of epidemic mumps through coughing, etc. The period of incubation seems to vary. On the average, it is about fourteen days.

Clinical History.—There may be a prodromal stage of one or two days, with mild feverish symptoms. The disease itself begins with swelling of one parotid gland. The swelling is directly below and in front of the lobe of the ear, which is gradually pushed upward. In the next few days the swelling rapidly increases, and it and the collateral œdema of the cheek and floor of the mouth may become very considerable. The face is much distorted, but often makes a very comical impression, especially as everybody knows how harmless the disease is. In most cases the other gland also swells a few days later.

Suppuration scarcely ever occurs in genuine mumps. If it is seen as a rare exception, it is probably due to secondary infection. The swelling often becomes very hard. Generally it has a somewhat doughy consistency. The corresponding portion of skin is usually pale and shiny. The submaxillary gland not infrequently swells also in addition to the parotid, and this may occur upon one or both sides of the neck. Penzoldt has observed cases in which the submaxillary and sublingual were swollen, but not the parotid. We ourselves have seen a swelling of the submaxillary precede the parotitis. The sublingual gland also seems sometimes to be involved with the parotid.

The local discomfort is moderate in most cases. There are local pain and difficulty in chewing, but less in swallowing and talking. Often quite a severe stomatitis develops, with foul breath.

There sometimes seems to be almost no fever. Usually there is a moderate elevation of temperature. Often, however, it reaches 102.5° to 104° F.

(39° to 40° C.). Only occasionally has there been a case with grave typhoidal symptoms. We have frequently observed herpes labialis in mumps. In rare cases a splenic enlargement can be demonstrated.

Complications.—It is not rare for men to have a swollen testicle with inflammatory serous exudation into the tunica vaginalis, which may be quite painful, but which usually subsides in a few days. The orchitis is usually ushered in with a fresh rise of fever. Resultant suppuration is a rare occurrence. Once we saw the orchitis appear several days before the swelling of the parotid gland. Double orchitis is rare. In boys this complication is much more exceptional than in adults. Some observers have mentioned analogous swellings of the female genitals (oöphoritis) and mammæ, but this is doubtful. Other complications, as facial paralysis, acute nephritis, pneumonia, etc., are rare.

Prognosis.—The prognosis of epidemic parotitis is, as we have said, almost always favorable. The trouble seldom lasts more than a week or ten days, when the swelling goes down, and the patient completely recovers. Possible but exceptional sequelæ are: ptyalism, cessation of the salivary secretion, chronic swelling of the parotid, deafness, and atrophy of the testicle.

Diagnosis.—The diagnosis is easy. The only thing to exclude is swelling of the lymph-glands, and they never have exactly the same location as the parotid.

Treatment.—Special treatment is hardly necessary. Children should be kept in bed. Usually some salve (lanolin) is applied to lessen the feeling of tension. If resolution is tedious, we may paint the swelling with iodized vasogen or with tincture of iodin; or we may prescribe iodoform ointment (1 to 15). If there is orchitis, the testicle must be elevated, as by a suspensory bandage. If the pain and swelling are marked, an ice bag should be applied.

2. SECONDARY SUPPURATIVE PAROTITIS

This secondary form may be a complication of any grave disease. In most cases it is due to inflammatory agents, probably staphylococci in most cases, generated by decomposition of the contents of the mouth, which agents reach the gland through Steno's duct. It was formerly the universal belief that the infection was metastatic, being conveyed through the blood vessels; but it is not certain whether this occurs. It is probable that the pyæmic form is in many instances thus produced. Secondary parotitis is most frequently observed in typhus and typhoid fevers. It is also seen occasionally in all other severe acute diseases, and in phthisis and carcinoma.

The parotid gland swells, just as in the primary disease. It is, however, much oftener of excessive size, and in the majority of cases suppurates. If one has an opportunity to make an autopsy on such a case of secondary parotitis in its early stages, the cross-section of the swollen gland presents a large number of rather small discrete abscesses. These finally unite to form one larger abscess, which usually discharges outward through the skin or into the external auditory meatus. Sometimes the parotid suffers from gangrenous inflammation, and there is often extensive sloughing. If such a case finally gets well, still, as a rule, some permanent injuries have been inflicted: there

is facial paralysis, due to destruction of the facial nerves, or deafness, caused by an extension of the inflammation to the middle ear.

The treatment of secondary parotitis is that of any suppurative inflammation. We may at first try to scatter the swelling by ice or a compress wet with alcohol, but this usually fails. As soon as fluctuation is detected, the spot must be incised, and a drainage-tube inserted. The prognosis depends chiefly on the nature and course of the original disease.

3. MIKULICZ'S DISEASE

This is a rare and still very obscure malady, which is characterized by a chronic swelling of the salivary glands, particularly the parotids, but also involving the submaxillary and the sublingual glands, and occasionally the lachrymal glands. In addition, there is usually a general anæmia. The patients very frequently develop tuberculosis later. Treatment has little effect and must be purely symptomatic. The attempt to reduce the swollen glands by regular X-ray treatment is to be recommended.

CHAPTER VIII

ANGINA LUDOVICI

The name *angina Ludovici* is applied to a rather rare phlegmonous inflammation of the floor of the mouth, first described by the Württemberg physician, Ludwig. Probably this is not occasioned by a specific cause, but is only a special localization of the ordinary germs which occasion suppuration. Its starting place seems to be the submaxillary gland, at least in most cases. It may be primary, or a complication of other severe acute diseases.

Angina Ludovici usually begins with swelling in the neighborhood of the submaxillary gland. The swelling rapidly increases, and comes to involve the whole floor of the mouth and the anterior surface of the throat. It causes great discomfort. Talking, chewing, and swallowing are almost impossible. There is usually fever, and sometimes we find the symptoms of general sepsis. There may be great dyspnœa, due either to compression of the larynx or to œdema of the glottis. The final result in some cases is an extensive sloughing of the soft parts. This has the special name of *cynanche gangrænosa*. In other cases an abscess forms, and points outward or into the oral cavity. The swelling is sometimes, though seldom, reabsorbed.

The prognosis should always be guarded, for severe constitutional symptoms and a fatal ending are not infrequently seen, particularly if the patient has a weakly constitution. There may also be repeated exacerbations and relapses.

Treatment.—At the commencement of the disease we may make the attempt, in suitable cases, to check the process by local depletion and by ice; but, as soon as suppuration or gangrene begins, the case becomes a surgical one. Now and then the threatening asphyxia demands tracheotomy.

CHAPTER IX

ANOMALIES OF DENTITION

(Difficult Dentition)

The processes of dentition play so important a rôle in the disorders of childhood that we feel obliged to discuss the subject, at least briefly.

The first appearance of any of the milk teeth usually takes place when the child is seven to nine months old; it may, however, occur either earlier (between the fourth and seventh months) or even later than this period. As a rule, the two lower central incisors are cut first; then the upper central incisors appear, a few weeks later, and next the lateral incisors of the upper jaw. In the beginning of the second year come the lower lateral incisors, and somewhat later the four anterior molars. The four canine, or "eye" and "stomach" teeth, are cut in the second half of the second year; and last of all comes the eruption of the four posterior molars. The first dentition is therefore completed by the end of the second or in the beginning of the third year, with the development of all the twenty milk teeth. The accompanying diagram (Fig. 69), after Vogel, represents the order in which the separate teeth appear. In the sixth or seventh year the milk teeth begin to be replaced by the permanent teeth of the second dentition. The milk teeth fall out in about the same order as they appear. "Trouble with teething," however, almost invariably refers to anomalies of the first dentition.

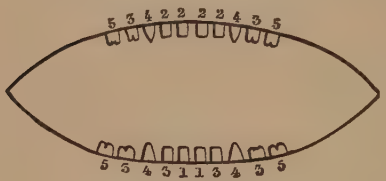


Fig. 69.

Noticeable delay in teething is frequent in constitutionally weakly, and particularly in rachitic children, in whom the teeth may develop not in pairs and in symmetrical groups, as in healthy children, but in irregular succession. In such cases, sometimes, all the teeth are not cut till the end of the third year.

On the other hand, it sometimes happens that certain teeth appear very early, or even are present at birth. If an abnormally early tooth is only loosely inserted in the gums, it should be removed with the forceps; for it interferes with nursing, and injures the opposing surface of the mouth; but if the tooth is firm in its place, we let it be.

During the eruption of the teeth there is in every child considerable redness of the mucous membrane and an increased flow of saliva. The child evidently feels an itching in the mouth, and therefore a constant desire to bite something. It is more restless, especially at night, and does not take food as well as usual. This simple catarrh is sometimes accompanied by a slight rise in temperature. Occasionally there is a severe stomatitis, with which fever and thrush may be associated. These troubles should be treated as already described.

In consequence of the salivation, and the large amount of saliva which is swallowed, in which the processes of decomposition are apt to develop, we often see gastro-intestinal diseases in teething children. In most children a temporary and mild diarrhea occurs. We should be particularly careful at

this period about the child's nourishment, and in treating any marked gastro-intestinal symptoms. Experience shows also that teething children are unusually liable to simple or even capillary bronchitis, and catarrhal pneumonia.

Nervous disturbances are often referred to dentition. The most important symptom of this kind is eclampsia. The attacks are sometimes called "teething convulsions." Although the laity go too far in ascribing all sorts of nervous disorders to teething, still experienced specialists do recognize the possibility of such an origin for many cases. Some of the convulsions may in fact be regarded as reflex (*vide infra* the chapter on the Convulsions of Children, Vol. II).

When the upper canines, or "eye teeth," are being cut, there is sometimes a unilateral purulent conjunctivitis, which is perhaps to be explained as an extension of the inflammation by way of the antrum of Highmore and the nostrils. Eczema and other cutaneous eruptions have been often ascribed to dentition, whether justly or not is doubtful. Yet it is remarkable that little children sometimes have an eruption of facial eczema with the appearance of every tooth.

Even healthy children frequently show a marked circumscribed hyperæmia of the cheek during the eruption of a tooth.

There is, of course, no special treatment for difficult dentition. Scarification of the gums to help the eruption of the teeth should never be practiced. The various disturbances appearing during dentition are to be treated on general principles.

SECTION II

Diseases of the Soft Palate, Tonsils, Pharynx, and Nasopharynx

CHAPTER I

VARIOUS FORMS OF SORE THROAT

(Inflammation of the Soft Palate and of the Tonsils)

ÆTIOLOGY

Acute inflammation of the soft palate and tonsils, in its various forms, is one of the commonest of diseases. Almost everybody has had personal experience with it. It is chiefly a disease of early life, being infrequent after the thirty-fifth year. Individual predisposition to it varies greatly. There are persons who have one or more attacks almost every year, while with others attacks are rare and insignificant. In many instances exciting causes have evidently been potent. Chief among these is catching cold; the sufferer has had wet feet, or has been talking in a damp, cold atmosphere. Most cases, therefore, occur in cool weather, although now and then attacks may occur in the hottest days of summer. Again, direct injuries of the pharynx may produce the disease—e. g., the smoky atmosphere of inns, combined with loud

talking or shouting; the inspiration of poisonous vapors; cauterization of the mucous membrane with concentrated acids, alkalies, and other chemical agents; and burns.

Apart, however, from these last-named external irritants we have many cases of primary angina, for which we must assume an infectious origin. This supposition is suggested by the severity of the constitutional disturbance, with fever, the nature of the local process (giving rise to suppuration), and the occasional appearance of the disease in an epidemic or endemic form. At the same time, exposure to cold and the other exciting causes above mentioned, may often favor its development. With regard to the nature of the infectious organism, if the tonsillitis is of the suppurative parenchymatous variety, it is certain that we have to deal with the ordinary pus organisms (usually staphylococci). The same is also true of follicular tonsillitis, although several different kinds of pathogenic organisms may be concerned; thus we may find not only staphylococci, streptococci, and particularly diplococci, but probably, in many cases, even the genuine diphtheria bacillus.

Frequently the inflammation is due to extension from neighboring parts, as in coryza, laryngitis, and stomatitis. In many cases both affections are simultaneous results of one common cause. Finally, sore throat may be a symptom of many acute infectious diseases, such as scarlet fever, measles, smallpox, and erysipelas.

To distinguish between an inflammation of the soft palate by itself and an affection of the tonsils is not practicable. In most cases the tonsils are the stronghold of the disease; less often we find the inflammation limited to the soft palate.

CLINICAL HISTORY

The most important subjective symptom of sore throat, and that by which it is usually first recognized, is the difficult and painful deglutition. The pain is sometimes manifest before any objective changes are to be seen. It may in a severe case be very violent and distressing. The pain has a "darting" character, or sometimes is "burning"; and it is most acute whenever the patient swallows, although in well-marked cases it seldom entirely intermits. Swallowing is not only painful, but it is laborious; it requires more than usual effort and time. The patient feels constantly as if he had to swallow a big lump. This sensation is worse if the tonsils are swollen. It is a matter of experience that not infrequently an "empty" swallowing hurts more than swallowing a liquid or some half-solid substance.

Talking is also difficult. Every word may be painful, so that the patient expresses his wishes as briefly as possible. Even in a mild case, speaking for any length of time will produce a burning pain in the throat. The impaired mobility of the soft palate often prevents the complete cutting off of the nasal passages in talking, so that the voice has a nasal twang; and it sounds as if the patient were talking with his mouth full: he has the "voice of sore throat."

Further local discomfort results from the mucus and saliva collecting in the mouth. Salivation is not infrequent, probably as a result of the stomatitis usually present. In other cases the patient complains that his mouth feels

dry and sticky. Frequently there is a persistent bad taste in the mouth, and the breath is disagreeable.

With these local disturbances, more or less severe constitutional symptoms are almost always conjoined. Indeed, these latter may begin a day or two earlier than the local symptoms. The patient is indisposed, languid, has anorexia and headache. The general disturbance may be surprisingly great in comparison with the slight objective changes in the tonsils.

There is fever in most of the well-marked cases; it may even be quite high. Temperatures of 103° or 104° F. (39.5° to 40° C.), or even higher, are not rare. Sore throat cannot be said to have one particular type of fever. Usually the fever appears rather abruptly, remains high for several days, with an occasional slight interruption, and then falls with equal abruptness to normal again.

The entire attack usually lasts only a few days, seldom more than a week. Even when a person is quite ill for several days, convalescence is almost always rapid and complete—that is, if the patient has a good constitution.

Special complications are rare, except that the neighboring parts—the larynx, mouth, and throat—are not seldom involved. The condition of the urine must be noted, as occasionally, after an apparently mild sore throat, an acute nephritis may develop. It is worthy of mention that herpes labialis is quite frequent.

VARIETIES OF SORE THROAT

The symptoms thus far mentioned are much the same in all cases of sore throat, varying only in intensity and duration. But the objective changes to be observed in the soft palate and tonsils are noticeably different in different cases. Whether the ætiology differs also we have no certain information. In some instances it seems probable that it does.

We shall distinguish five chief varieties of acute sore throat. Transitional forms are, however, by no means rare. Genuine diphtheria, which is a specific, acute, infectious disease, and which has already been discussed, does not need to be brought up again here.

1. **Catarrhal Sore Throat** (*Simple Catarrhal Inflammation of the Mucous Membrane of the Soft Palate*).—There is a more or less vivid reddening of the mucous membrane, either uniform or in patches. The swelling is most marked in the pillars of the fauces and the uvula. The surface of the tonsils is likewise reddened; their size may be somewhat increased, or remain unchanged. The mucous membrane of the palate and uvula may be covered here and there with a thin layer of mucopus, which can easily be wiped off. The tonsils may present small superficial erosions scattered about. These little ulcers are apt to lie at the openings of the follicles. The small "blisters" which are often seen on the mucous membrane of the soft palate are caused in various ways. Either they are mucous glands or solitary follicles, swollen; or, rarely, they are real vesicles filled with a clear fluid and produced by a raising up of the epithelium. The cervical lymph-glands are usually but slightly swollen if at all.

This is the common and mildest form of sore throat; and it may be over in a day or two. In some instances, however, it causes considerable local

and general discomfort; but the disease seldom lasts longer than five to eight days.

2. **Follicular Tonsillitis.**—In this frequent and practically important form there is not only more or less catarrhal inflammation of the soft palate, but a decided swelling of one or both tonsils. On the reddened surface of these swollen bodies are whitish-yellow spots, varying in number from two or three to ten or more, and corresponding to the follicles. These spots are often seen to be plugs projecting from the openings of the follicles. It is usually easy to press out the pasty contents of the follicle, represented by the white speck, with a spatula. The microscope shows it to consist of numerous epithelial cells and pus corpuscles, bacteria, and detritus, and sometimes there are also crystals of the fat acids and cholesterin. The pus corpuscles may so predominate that we may have small follicular abscesses, which, on being opened, leave superficial ulcers behind. The parenchyma of the tonsil is swollen with a serous and cellular infiltration, increasing the bulk of the part. The trouble is usually bilateral, though often more marked and extensive on one side than on the other. In the severer cases the cervical lymph-glands are usually moderately swollen.

The local symptoms and still more the constitutional disturbance in follicular tonsillitis may be decided. The fever not infrequently reaches 104° F. (40° C.) or even higher. The patient feels very languid, has no appetite, and complains of headache; but there are mild as well as severe cases. The disease never lasts longer than a few days, even when the fever is high at first. The yellow spots vanish from the tonsils, and there is rapid recovery; severe complications are hardly ever seen. Often there is a herpes labialis and there may be slight albuminuria, but this very rarely amounts to a genuine acute nephritis. It should be mentioned that sometimes the contents of a few of the follicles remain in them for a considerable length of time, and become inspissated and calcified. It is not a rare thing to find such plugs in the tonsils of those who are subject to sore throat. Timid and hypochondriacal individuals are sometimes badly frightened by expectorating these old chalky plugs, which they believe to be "tubercles"!

From an ætiological standpoint, follicular tonsillitis is in most cases entirely distinct from genuine diphtheria, and so far the search for the Klebs-Löffler bacillus in the plugs contained in the follicles has almost invariably proved negative. Still, it is noteworthy that precisely at the time of diphtheria epidemics follicular tonsillitis is remarkably frequent. It is also bacteriologically determined that the mildest degrees of diphtheria present themselves in the garb of an apparently simple follicular tonsillitis.

3. **Tonsillar and Peritonsillar Abscess** (*Parenchymatous Sore Throat; Quinsy*).—In this form the swelling of the tonsils and the surrounding tissues is the most striking symptom. They may be more than twice their natural size. The anterior pillars of the fauces are pushed forward and become convex. The swelling extends so far toward the median line that the tonsil touches the uvula; or, if the affection is bilateral, the two tonsils press against each other, grasping the uvula between them, or pushing it forward. The soft palate is very much reddened, particularly at first. Its surface is usually thickly covered with mucus. If this be wiped off, the mucous membrane is seen to have a moist, œdematous luster. The mucous membrane of the tonsils

not infrequently suffers a superficial necrosis. Follicular and parenchymatous tonsillitis are often combined.

In well-marked cases of abscess the local discomfort is usually great. With every attempt to swallow there is pain which shoots into the ear. The patient is in a pitiable condition; he can neither talk, nor swallow, nor gargle. The few words which he painfully utters have in an extreme degree the nasal quality of the "voice of sore throat."

In the milder cases the trouble seldom lasts but a few days before the swelling goes down, and the discomfort and usually rather moderate fever gradually abate. In other cases an abscess of the tonsil develops, usually on one side only, but it would be more correct to say a peritonsillar abscess, for the pus usually collects mainly in the connective tissue between the tonsil and the arch of the palate. In such cases the mucous membrane bulges out, usually at a point in the velum of the palate, and on palpation there is a sense of fluctuation, and finally the abscess ruptures. In other cases, however, a tonsillar abscess forms, as a rule, on only one side. The mucous membrane bulges out more in one spot; distinct fluctuation is detected; and, finally, the abscess breaks. With the discharge of the pus the pain is relieved very rapidly, or it may vanish at once. The rest of the tonsil soon regains its former size, and in a few days the patient is well. Relapses are possible, but rare.

Phlegmonous sore throat, in which the soft palate and not the tonsil is chiefly affected, is infrequent. Its usual cause is some severe external injury, such as burns, and cauterization with concentrated acids or alkalies. The swelling extends deep down into the submucous tissue. The uvula may have the diameter of one's finger. There is intense hyperæmia. Sometimes there are hemorrhages into the mucous membrane: this is called hemorrhagic sore throat.[1]

4. **Necrotic Tonsillitis** (*Necrotic Sore Throat*).—This name is applied to a disease which is not infrequent, and in which the tonsils are the main seat of the pathological processes. The pillars of the fauces and the uvula are but slightly affected with a simple catarrhal inflammation. The tonsils are, as a rule, moderately swollen, seldom attaining great size. The mucous membrane covering them presents a whitish or grayish-white discoloration, often quite extensive, and most marked on the side toward the uvula. A careful investigation shows that there is in reality no "coating," but a necrosis. The process may be superficial; sometimes it reaches quite deeply into the structure of the mucous membrane. It is not possible to pull off this white matter, as one can loosen croupous membranes, although little bits may perhaps be scratched off with a spatula or a pair of forceps. These particles are found, on microscopic examination, to be made up of detritus, bacteria, epithelium, and pus corpuscles. The necrosis is almost invariably confined to the tonsils, and a sharp boundary line separates it from the reddened and inflamed pillars of the fauces. After a few days the slough may come away, leaving behind an ulcer, which, though usually shallow, has sometimes a considerable depth. This generally cleans up rapidly. In severe cases, however, the floor of the

[1] Another form of hemorrhagic sore throat occurs where there is violent tonsillitis with necrosis or gangrene. There is also a necrotic, hemorrhagic sore throat accompanying scurvy and leukæmia.

ulcer consists for a number of days of a dirty necrotic material, which comes away only gradually. The worst cases may be properly called "gangrenous tonsillitis."

Necrotic tonsillitis is almost always attended by considerable fever and marked constitutional disturbance. Children particularly seem very ill in the first days of the attack. The cervical glands are usually swollen, but seldom as much so as in genuine diphtheria.

Despite the rather ominous commencement, the disease does not last a great deal longer than the other forms of sore throat. It seldom continues more than five to eight days before a speedy convalescence begins.

The necrotic tonsillitis is distinguished from the follicular form by the greater area of the white or grayish-white spots. Still, it should be particularly noted that sometimes combinations of these two varieties, or transitional forms, occur.

Only the bacteriological examination can decide with certainty the ætiology of necrotic tonsillitis. In some cases diphtheria bacilli can be demonstrated. Then, of course, the infection is a true diphtheria which presents the anatomical picture of a necrotic tonsillitis. In other cases there are present no diphtheria bacilli, but only the ordinary inflammatory organisms, particularly streptococci.

At this point we wish to refer briefly to the so-called *Plaut-Vincent's Angina,* a form of tonsillitis first described by Plaut and Vincent in 1898, and which in many respects resembles diphtheria. A grayish-white coating develops on one or both tonsils; after this separates there remains a superficial erosion of the mucous membrane, or occasionally a deeper ulcer (ulcero-membranous tonsillitis). The constitutional manifestations (fever, glandular swellings, joint pains, albuminuria, etc.) may be quite severe. The disease runs its course in about one to one and one half weeks. The bacteriological examination shows no diphtheria bacilli but generally the so-called *Bacillus fusiformis,* together with numerous spirillæ and other bacteria. Of late, small epidemics of this form of tonsillitis have been repeatedly observed.

DIAGNOSIS AND PROGNOSIS

It is never very difficult to recognize a sore throat, and a little practice makes it easy in most cases to decide what particular variety is present, if we examine the objective changes carefully. It is very important in practice to distinguish diphtheria from the benign forms of inflammation. Follicular and necrotic tonsillitis are very frequently mistaken for diphtheria—an error which explains the success of a large number of remedies said to cure diphtheria. Many physicians call every case of sore throat, where there is anything white to be seen, "diphtheria." Certainty in diagnosis of genuine diphtheria can be gained only by practice; no description, however complete, can take the place of personal observation. It may be a help to remember that in both follicular and necrotic tonsillitis the white spots are usually limited to the tonsils, while in croupous sore throat the deposits are generally from the very first also situated upon the pillars of the fauces and the uvula. The white spots of the follicular variety can generally be at once recognized by their arrangement. The plugs and areas of suppuration are seen pro-

jecting from their follicles. In necrotic sore throat there is never a separable croupous membrane with its characteristic histological structure, but there is simply a superficial necrosis of the mucous membrane and parenchyma. The condition of the cervical lymph-glands is not unimportant; as a rule, they are much more affected in diphtheria than in the benign cases.

It is further to be noted that the fever in true diphtheria rarely reaches the point that it does in the tonsillitis produced by pus organisms. A tonsillitis that begins at once with fever of about 104° F. is rarely a true diphtheria.

Still, the fact must again and again be emphasized that only the bacteriological examination can definitely decide each individual case. It certainly is worth while, especially with children, to isolate all cases that are in any way doubtful.

TREATMENT

These troubles usually run so favorable a course that active treatment is very seldom needed. The gargle usually prescribed generally gives the patient more discomfort than relief. The most common prescriptions are: Solutions of potassium chlorate (10 to 300) diluted with lukewarm water, peroxid of hydrogen, etc. To paint the parts is a useless and now almost obsolete proceeding. Inhalations of spray are better, with alkaline solutions or tannin. The use of formamint tablets is also serviceable. They are allowed to dissolve slowly in the mouth. It is beneficial to put a cold or hot wet compress around the throat. Children must be kept in bed, and adults are generally forced to go to bed, if the constitutional symptoms are well marked. If the febrile symptoms are marked, antipyretics (antipyrin, phenacetin, and aspirin) sometimes afford relief.

We try, unfortunately often without result, to relieve the symptoms of parenchymatous tonsillitis with ice pellets or warm mouth washes. If there is evident fluctuation, we can make an incision with a spear-pointed bistoury, after guarding a portion of the blade with sticking plaster. The best place is almost always in the velum palati, corresponding to the outer boundary of the tonsil. Great relief follows; and even if there is no distinct abscess an incision will usually give relief, if there is excessive swelling, and if there is a deeply seated abscess will facilitate its spontaneous discharge. Punctures of this sort cause scarcely any pain.

[Hoffman has pointed out that in some cases permanent recovery can be attained by breaking down the partitions between the crypts of the tonsils, thus establishing thorough discharge of their secretions.]

CHAPTER II

CHRONIC HYPERTROPHY OF THE TONSILS

Chronic hypertrophy of the tonsils occurs not only in those who have had repeated attacks of tonsillitis, but also in cases in which no occasion for it can

be found. Even in childhood there is not infrequently a well-marked hypertrophy, which must be due to a special congenital predisposition.

The condition is at once revealed by inspection. There may be no signs whatever of any acute or chronic inflammation, or there may be an accompanying chronic pharyngitis. The tonsils bulge out in two great lumps. They may be so large as to touch the uvula on each side. Histologically, there is a genuine hypertrophy of the organ—that is, an increase of all its component tissues.

In many cases, where the swelling is moderate, there is no discomfort. The possessor of the tonsils is not aware that they are enlarged. In other cases the hypertrophy proves of clinical importance, inasmuch as all forms of sore throat are found to occur more frequently if the tonsils are enlarged, and to cause more trouble when they do appear. The hypertrophied organs may also be the seat of a chronic catarrh, which by extension gives rise to chronic nasal catarrh, catarrh of the Eustachian tubes, or hoarseness.

If the hypertrophy is considerable, the local discomfort may be quite marked. Swallowing is rendered difficult, if not painful. Frequently there is evident dyspnœa. The patient has to breathe through his mouth, and sometimes when asleep snores and snorts in a way to frighten one. Children are particularly apt to suffer in this manner. Many instances of *pavor nocturnus,* or "night terrors," in children are referable to this cause. We have already mentioned that cases of bronchial asthma sometimes seem to be connected with hypertrophy of the tonsils.

Treatment.—The attempt to reduce the enlargement by applying lunar caustic, tincture of iodin, etc., usually fails. If there is much distress, if the patient is subject to frequent sore throats, or if the hypertrophy of the tonsils keeps up a chronic nasal or pharyngeal catarrh, then the only remedy is to remove the tonsils by operation. The operation should not be undertaken, however, without sufficient reason.

CHAPTER III

CHRONIC PHARYNGITIS

ÆTIOLOGY

It is not practicable to distinguish between chronic catarrh of the soft palate and of the pharynx, for, as a rule, the two are combined. Sometimes the condition is the result of repeated acute attacks; sometimes—and probably oftener—it is due to persistent, injurious, local influences. A large number of cases originate in bad habits, or in abuse incident to certain vocations. Examples are seen in smokers, drunkards, singers, preachers, teachers, and men who work outdoors. In talking and singing, the soft palate is strained; or the disease is excited by breathing cold or impure air, or by such chemical irritants as alcohol or tobacco. In many cases, chronic pharyngitis follows chronic rhinitis or chronic laryngitis. The general passive congestion due to cardiac disease or pulmonary emphysema may sometimes promote the development, or prolong the existence, of a chronic pharyngitis.

SYMPTOMS

The local discomfort is often slight. The patient gets used to it, and does not mind it except when there is some exacerbation. It becomes a more important matter if the calling of the patient is interfered with, as in a preacher, singer, or teacher.

Deglutition is seldom impaired. There is often, however, a constant feeling of dryness, burning, or scratching in the throat. The patient has to clear his throat frequently, and often acquires an habitual, short, sudden cough, which may or may not be dry. The expectoration, if there is any, is almost pure mucus. When there are violent efforts to clear the throat, there may be a slight hemorrhage from the dilated blood vessels of the posterior wall of the pharynx, explaining the presence of small amounts of blood in the expectoration. This blood often has a dried-up look, as if not fresh. It sometimes leads overanxious patients and physicians to suspect pulmonary hemorrhage. The uvula becomes so long that its tip rests on the tongue or the posterior wall of the pharynx; and this gives rise to a peculiar and disagreeable sensation of tickling. All these uncomfortable feelings are temporarily increased if anything affects the throat unfavorably; and they are generally at their worst on rising in the morning, apparently because the mucous membrane has become dry, or a collection of tough mucus has formed during the night. Everyone knows how drunkards have to hawk and cough in the morning, so that often they almost strangle or vomit.

On inspection, we generally find the mucous membrane reddened. Very often a number of dilated and tortuous veins are visible both on the soft palate and in the back of the throat. Of equal frequency is the appearance of numerous small gray projections, corresponding usually to swollen follicles or else to hypertrophied mucous glands. This is called granular pharyngitis. Small follicular ulcers are not infrequent. Exceptionally there are more extensive catarrhal ulcers. The mucous membrane of the posterior wall of the pharynx may present patches of opaque or thickened epithelium, giving the surface a grayish-white appearance.

Frequently chronic pharyngitis is combined with chronic laryngitis, evidenced by hoarseness; or with posterior nasal catarrh, or catarrh of the Eustachian tube, producing deafness and ringing in the ears.

VARIETIES OF CHRONIC PHARYNGITIS

1. **Chronic Catarrh of the Nasopharynx, or Chronic Posterior Nasal Catarrh.**—This has the same ætiology as the ordinary form. It is practically important because the nose and ear are frequently involved.

The anatomical changes are essentially those already depicted under chronic pharyngitis. The region affected cannot be seen by direct inspection, so that accuracy in diagnosis requires the use of a nasal speculum (see particulars in the works mentioned on page 164). The ordinary examination of the throat may reveal a condition which is quite characteristic of posterior nasal catarrh: a collection of mucopus, or of firmly adherent dry crusts, is seen on the posterior wall of the pharynx, and extends upward toward the nasopharynx.

The local discomfort is somewhat similar to that experienced in chronic pharyngitis. There is a scratchy feeling, or a feeling as if there were a foreign body in the back of the throat, accompanied by a constant desire to blow the nose, hawk, or cough. Dried and decomposing secretion often causes extremely foul breath. There is often also vertigo and occipital headache.

In many cases the nostrils are stopped up. The posterior opening of the nostrils is closed in part by the swelling and hypertrophy of the mucous membrane, and in part by the accumulated secretions. The patient, therefore, usually has to breathe through the mouth. The ear is frequently involved. The catarrh extends into the Eustachian tubes and the tympanic cavity, or the opening of the tubes is occluded with the secretions. For a detailed consideration of the deafness, tinnitus, etc., thus produced, consult works on otology.

2. **Pharyngitis Sicca,** or *"Dry Atrophic Catarrh of the Throat and Nasopharynx."*—This name is applied to an atrophic disease of the mucous membrane, which sometimes is spontaneous and sometimes is a sequel of chronic pharyngitis. The whole mucous membrane of the pharynx and the nasopharynx (seen with the rhinoscope) seems pale, smooth, and perfectly dry, and has a peculiar luster, as if varnished. Here and there tortuous veins project from the general anæmic surface.

If an opportunity is afforded to examine the mucous membrane microscopically, it will be found that the atrophy involves all the elements of the tissue, though the follicles and mucous glands suffer most.

This condition may not cause any symptoms, but, in many cases, the patient suffers considerably. The chief trouble is a feeling of dryness in the throat, rendering deglutition difficult or even painful. There is also a constant desire to clear the throat. The secretion hawked up may be scanty and tough or more abundant, and it is often tinged with blood. Actual coughing may also be due to pharyngeal trouble ("throat cough"). Talking is often rendered difficult, the voice grows weak, and it becomes easily tired. In severe cases there is also general debility. Not infrequently pharyngitis sicca is associated with atrophic rhinitis (*q. v.*), but it occurs also where there is no nasal disease.

The disease is most frequently seen in the elderly, but it also occurs in children and young persons. It is especially common in ill-nourished individuals, or in those who are suffering from such diseases as tuberculosis or chronic nephritis.

3. **Hypertrophic Catarrh in the Pharynx and Nasopharynx.**—An opposite condition of hypertrophy sometimes results from chronic catarrh. The changes consist mainly in hyperplasia of the lymphatic tissue, and they are usually termed "adenoid growths." The choanæ and the posterior extremity of the nasal septum may be almost completely hidden by these growths, as they extend down from the roof of the pharynx in grayish-red, uneven masses. In the majority of cases the hypertrophy seems to originate chiefly in Kölliker's "pharyngeal tonsil."

The adenoid growths are especially common in childhood. The symptoms consist in a change of the voice (which loses its reverberating quality and becomes nasal), frequent snuffling and hawking, and a tough mucous secretion.

often tinged with blood. Not infrequently there is headache. Of greater importance is ear trouble, which is often occasioned by the growths.

An accurate diagnosis requires rhinoscopy. Positive results are also often obtained by palpation. The index finger, being pressed backward and bent upward, can touch the protuberances and the enlarged pharyngeal tonsil in the nasopharynx.

PROGNOSIS

In all varieties of chronic pharyngeal catarrh, the prognosis as to recovery is doubtful, for in all severe cases the process is very obstinate, and permanent restoration to health exceptional. It is essential for the best results that all injurious influences should be removed. Even if decided improvement occurs there is a tendency for the trouble to grow worse again, and for acute exacerbrations to occur.

TREATMENT

Many of the milder cases never apply to a physician. The patient uses some domestic remedy or gargle, or becomes so accustomed to the disagreeable sensations that he does not consider it necessary to do anything in particular about them.

The treatment of a well-developed case requires great patience and persistence on the part of all concerned. If there is some underlying disease, such as pulmonary or cardiac disease, that must be treated. All exciting causes must be avoided. Energetic local treatment is also indispensable. I cannot, however, refrain from calling attention to the fact that the local treatment can also be overdone. Not very infrequently patients with chronic pharyngeal catarrh are painted or cauterized locally for months without any particular benefit. Such a long-drawn-out treatment is, particularly in worrisome neurasthenics, sometimes more harmful than beneficial. Regarding the methods of local treatment, the following remarks will meet the requirements of ordinary practice:

Gargles are seldom satisfactory, for they never reach farther than the soft palate. Inhalations are better; we may use solutions of alum or tannin, or, in mild cases, of common salt. Still more efficient is the painting of the entire surface of the pharynx with some concentrated solution. The physician usually has to perform this, although some patients learn to do it for themselves. Proper solutions are: Argentium nitrate, five or ten per cent; tannin, eight to twenty per cent; tincture of iodin, either pure or diluted; or iodized glycerin, composed of pure iodin, parts 0.5; iodid of potassium, 2; glycerin 20, with the addition, if desired, of two drops of oil of peppermint. These applications must reach all the diseased surface. If the nasopharynx is involved, the brush must accordingly be bent upward, to reach that region. For this a mirror may be needed. It is very important to make the applications to the mucous membrane itself, freed from any interposing secretions.

In the treatment of chronic posterior nasal catarrh the nasal douche (*vide* diseases of the nose) plays an important part. It should be used two or three times a day. It not only removes the collected secretions, but it is a means of making local applications. The instrument is merely a fountain syringe.

The nozzle must be of a size to fill the nostril completely. The force of the current should always be moderate, and the patient's head should be sharply flexed forward. The fluid used—the best is a one-per-cent solution of sodic chlorid or bicarbonate—must have about the temperature of the body. Other medicated solutions must be very weak, such as sulphate of zinc, 1 to 1.000.

The insufflation of powders into the throat can be made through any small glass tube three to six times a week. Alum or tannin may be used, either pure or mixed with equal parts of pulvis gummosus [P. G., made of gum arabic, 3 parts; licorice root, 2 parts; and sugar, 1 part], or 3 to 5 parts of nitrate of silver to 100 of starch. For the nasopharynx, a bent tube of glass or hard rubber is to be introduced through the mouth. There are numerous "insufflators" to be had at the instrument makers.

Many baths enjoy a great reputation for the cure of chronic pharyngitis. Besides Ems, there are Reichenhall, Kreuznach, Salzungen; the cold sulphur springs, such as Weilbach, and many others. Good results are also achieved in Kissingen and Marienbad, if these places are favorable to the patient's general constitution.

In pharyngitis sicca, the nasal douche with a one-per-cent salt solution is to be recommended. It is sometimes also beneficial to paint the parts with solution of argentic nitrate, iodized glycerin, etc. Many irritating influences which do harm in common pharyngitis seem sometimes actually to benefit this form—such as smoking and taking snuff.

In treating the hypertrophic forms of pharyngitis and the adenoid growths, cauterization with nitrate of silver suffices for the milder cases alone. A thorough and permanent cure can be achieved only by removing the growths through operation.

[To Hooper, of Boston, belongs the credit of showing that a radical operation for the removal of adenoid growths of the nasopharynx can safely be done under anæsthesia. The blood does not, as was feared, run into the air-passages, but either into the stomach or outwardly. Anæsthesia thus renders it possible to scrape away with the finger or tear away with forceps the hypertrophied tissue at one sitting, and to do it in the child before secondary changes have taken place in the facial expression, the jaw, the ears, or the form of the chest.]

CHAPTER IV

RETROPHARYNGEAL ABSCESS

Retropharyngeal abscess is formed by a suppurative inflammation of the connective tissue lying between the posterior wall of the pharynx and the spinal column. It is a serious disease, although a rare one. If unrecognized, it proves fatal in many instances; while, if a correct and timely diagnosis is made, the patient can usually be easily cured. It is commonest in childhood, and before the second year. It almost always appears as a primary, acute disease, without any special cause being evident. Probably the agents which excite the inflammation penetrate into the tissue from the pharynx. The idea that the inflammation originates in the small lymphatic glands which lie in front of the vertebræ lacks proof as yet.

The disease attacks not only weakly children, but those who have been perfectly healthy and vigorous. The child grows restless and fretful, and does not nurse well. Apparently, deglutition soon becomes painful, but one cannot be certain about this except in older children. Generally, the respiration quickly takes on a peculiar stertorous character, particularly during sleep. Mucus collects in the mouth and throat. Upon swallowing there is often regurgitation through the mouth or nose, or some of the food gets into the windpipe and causes violent coughing. The lymph-glands of the jaws are usually somewhat swollen, and the neighboring parts may seem slightly œdematous. After a week or two the dyspnœa gradually increases. Respiration becomes more and more laborious, with loud rattling, and signs of stenosis. The jugular veins become distended, the lips cyanotic, and portions of the thorax are retracted during inspiration. The voice is feeble, and may be hoarse and indistinct.

The correct interpretation of these symptoms, which are common to various disorders, requires a careful examination of the throat. It must be confessed that this has its difficulties in an infant. Still, we can sometimes see distinctly a swelling in the posterior wall of the pharynx. This may be either in the median line or on one side. All doubt is removed by digital examination, in making which, however, we must insert a wedge between the teeth, to avoid being bitten. The finger detects fluctuation.

The diagnosis once established, the abscess must be opened at once. We should not delay, even if the dyspnœa has not yet become extreme. To use the finger nail for the purpose, as has been recommended, is permissible only in an emergency. As a rule, incision is made with a bistoury, of which all but the point is guarded with sticking plaster. The left index finger is placed upon the abscess, and used as a guide. Meanwhile, the child's head is kept upright, and, as soon as the cut is made, bent over forward. The pus pours out in abundance. It is advisable to syringe out the mouth repeatedly with lukewarm water. The threatening symptoms vanish almost instantly upon the escape of the pus. Exceptionally, the abscess refills and requires a second incision.

If the trouble is not correctly diagnosticated, or if the abscess is not opened promptly, the patient may suffocate. The abscess, however, may burst spontaneously; then there is either speedy recovery or asphyxia from the pus filling the larynx. In some instances, when a retropharyngeal abscess has not been properly treated, the pus has gravitated far down into the neck and posterior mediastinum. The recognition and incision of the abscess may prove very difficult if from the start it is situated lower down in the throat than usual.

Analogous to this acute idiopathic abscess of which we have been speaking is the chronic abscess due to caries of the cervical vertebræ. This occurs in children as well as adults. Incision of such abscesses is indicated only when there is danger of suffocation. Because of their tuberculous character, the prognosis is, of course, much more unfavorable than in ordinary suppurative inflammation.

Retropharyngeal abscesses sometimes occur in pyæmia or other severe acute infectious diseases, but they have hardly any interest except to the pathologist.[1]

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[1] Tuberculosis of the pharynx is spoken of in the chapter on Pulmonary Tuberculosis. New growths in the mouth or pharynx belong to the domain of surgery.

SECTION III

Diseases of the Œsophagus

CHAPTER I

INFLAMMATION AND ULCER OF THE ŒSOPHAGUS

Ætiology and Pathology.—The various forms of œsophageal inflammation and ulceration are not of very great clinical importance. The processes are seldom of a severe grade, or, if so, they are generally a part of some complicated disease, to which they seldom contribute prominent symptoms. Very likely the milder forms of inflammation occur frequently, but the symptoms are hardly ever characteristic.

A simple catarrhal inflammation of the œsophageal mucous membrane may be caused by swallowing substances which are injurious mechanically, chemically, or from their temperature. It may also occur in the general infectious diseases, such as typhoid and typhus fevers, and the acute exanthemata. Any inflammation of neighboring tissues may extend into the œsophagus. Chronic catarrh is seen in heart disease, from the passive congestion. It is also found in the vicinity of other chronic œsophageal diseases, particularly cancers and diverticula (*vide infra*).

The acute catarrh is distinguished by not having the usual increase of secretion. The epithelium grows spongy, as a rule, and is cast off more rapidly than usual, so as to suggest the name of a desquamative catarrh. It is in only a few cases that the scanty mucous glands become swollen and look like papules upon the surface of the membrane; this form is called follicular catarrh. In limited areas the desquamation may be complete, giving rise to small catarrhal erosions. Likewise, the swollen follicles may break down into small follicular ulcers.

In chronic catarrh there is a moderate increase in the secretion of mucus, and a marked thickening of the epithelium. In very protracted cases actual papillomata may finally be formed. In some cases ulcers are seen.

Croupous and diphtheritic inflammations of the œsophagus are very rare. We have already said that the specific pharyngeal diphtheria frequently extends into the larynx, but only exceptionally into the gullet. Still, we have ourselves seen in children several cases of cicatricial œsophageal strictures (*vide infra*) subsequent to an attack of diphtheria. Isolated cases of diphtheritic œsophagitis have also been seen in connection with severe infectious diseases, such as typhus, typhoid, smallpox, cholera, pyæmia, and pulmonary tuberculosis, as well as in the course of Bright's disease and cancer. In variola it is not unusual for pocks to appear upon the œsophageal mucous membrane.

A purulent, phlegmonous œsophagitis now and then attacks the submucous layer. It may be either diffuse or circumscribed. The mucous membrane is dissected up from the muscular layer by the pus, and pushed inward, so as to diminish the lumen of the œsophagus more or less. Most of the cases end by the discharge of pus into the tube, when complete recovery may ensue. If the mucous membrane, however, has been extensively undermined, Zenker

states that a fissure-like cavity may be left even after healing has taken place. Its walls grow smooth, and finally acquire a layer of fresh epithelium.

Purulent œsophagitis is caused either by the presence of foreign bodies in the œsophagus, or by purulent inflammation in neighboring parts, as in glandular abscess, vertebral abscess, or laryngeal perichondritis. It has now and then resulted from the action of concentrated acids and the like upon the mucous membrane.

The action of corrosive poisons (corrosive œsophagitis) is to cause necrosis of the tissues, which in its turn produces inflammation. The inner surface of the œsophagus is converted into a rotten, hemorrhagic, sloughing mass, of a dirty gray or almost black color. In severe cases the muscular layer itself may be partly destroyed. If death does not occur speedily, the necrosed portions come away, leaving extensive purulent ulcers behind. These, if they heal at all, cause large cicatrices and stenosis.

Symptoms.—The milder cases, as we have stated, produce almost no distinctive symptoms. Possibly there may be pain along the œsophagus, or at some one point in it, during deglutition. In a more severe case the pain may be great; but the other symptoms are usually too grave for this to excite special attention. Laborious deglutition, and the feeling as if the food were inclined to stick in the throat, result from implication of the muscular layer. A diagnosis of the particular form of œsophagitis is attainable only when the ætiology guides us to it.

Treatment must be purely symptomatic. No solid food should be taken. The pain is to be allayed by bits of ice or by morphin.

CHAPTER II

DILATATION OF THE ŒSOPHAGUS

1. DIFFUSE DILATATION

Diffuse, spindle-shaped dilatation of the œsophagus is observed as a result of organic stricture of the cardiac orifice. At first the muscular coat hypertrophies as the orifice contracts, and is able to overcome the obstruction, so that there is no dilatation; but as soon as the muscles are paralyzed, and food collects behind the stricture, the dilatation begins and keeps on increasing. The ectasis is greatest at the lower end of the tube, as is natural from its mode of origin, and gradually diminishes upward.

There have been a very few well-substantiated instances of this diffuse spindle-shaped dilatation, without any demonstrable anatomical stenosis of the cardiac orifice. Their mode of origin is still doubtful. At present most writers assume that in these cases there exists a constant abnormal cramp of the cardia (cardiospasm). As yet there has been no certain proof of this hypothesis. In favor of the assumption of a cardiospasm is the observation that the bougie in these cases can sometimes be easily passed into the stomach, while at other times this is impossible. This phenomenon can perhaps be explained by other kinds of mechanical disturbance at the lower end of the œsophagus, such as kinking, pocket formation, etc. Possibly the chief factor

is a congenital deviation in the formation and growth of the œsophagus, such as the development of a so-called "*proventriculus.*" The mucous membrane of the œsophagus is usually but slightly changed in these cases, but the muscularis is as much hypertrophied as in genuine stenosis of the cardia.

Actual Cardiac Strictures.—If an actual stenosis of the cardia from cicatrix or carcinoma is present, this, and not the secondary dilatation of the œsophagus is, of course, the real cause of the existing dysphagia and the subsequent regurgitation of any ingested food. The patients themselves often feel that food sticks in the gullet, and that a large part of it does not reach the stomach at all.

It is much more difficult to explain the symptoms in diffuse dilatations of the œsophagus without demonstrable macroscopic changes at the cardia. The malady develops very gradually. At first the patients complain only of indefinite painful sensations at the sternum. Gradually, however, difficulty in swallowing supervenes; the food taken does not reach the stomach at all, or only a very small part of it does. Very soon it is regurgitated. Sometimes it remains for a considerable time in the dilated œsophagus, and is expelled only by straining and vomiting. In such cases, the œsophageal disease is sometimes confounded with a disease of the stomach (ulcer, pyloric stenosis, etc.), which error in diagnosis may lead to incorrect therapy. The diagnosis is possible only when the results obtained by using the sound and by washing out the œsophagus and stomach are carefully considered. In difficult cases, œsophagoscopy and the X-ray examination of the œsophagus, filled with a mixture of bismuth and mashed potato, must be resorted to.

As soon as the nutrition is permanently disturbed, there is loss in weight and strength, which may finally reach an extreme stage. The problem facing the physician is to maintain the nutrition by means of the stomach tube. If it is possible to introduce the tube into the stomach, then the condition of the patient will be rapidly improved through the newly added nourishment. If for any reason the introduction of the tube into the stomach is impossible, all that remains to be done is rectal feeding (*vide infra*) (which, after all, is not sufficient for any length of time), or the establishment of a gastric fistula. Upon the success of this operation and the nature of the primary lesion depends the ultimate outcome of the disease. In the so-called cardiospasm, the artificial dilatation of the cardia has been repeatedly practiced with good result. A collapsed, small rubber balloon is introduced into the cardia, and gradually inflated with air. All these mechanical methods of treatment require great care, if we do not wish to experience unfortunate consequences, such as secondary inflammation, and even rupture of the œsophagus, which I know to have occurred in one case. [See also remarks on page 339.]

2. DIVERTICULA

Ætiology and Pathology.—Circumscribed pouches in the wall of the œsophagus are termed diverticula. They are divided into two essentially distinct varieties, according to their mode of origin. Zenker has given them the names of pressure and traction diverticula.

The diverticulum due to pressure is extremely rare. It is caused by pressure upon the mucous membrane from within, by which some abnormally

weak spot is forced outward. All cases that have been carefully examined thus far have shown that, histologically, the wall of the diverticulum is not the distended but otherwise unchanged wall of the œsophagus, but is composed exclusively of the mucous membrane and the thickened submucous coat. We are therefore obliged to suppose that the mucous membrane is pushed out like a hernia through some gap in the muscular coat. It is only about the neck of the diverticulum that any muscular fibers are found.

The original factor, therefore, in the occurrence of a pressure diverticulum is apparently to be sought in some circumscribed lesion of the muscular coat. As a result of several observations, it is established that a foreign body, sticking in the throat, may separate some of the muscular fibers and push the mucous membrane through the gap thus formed. Or a severe injury leads to a trifling rupture of the muscular coat, and then the food, as it is being swallowed, presses out the mucous membrane at this weakened spot. There are still many other cases in which the true origin of the diverticulum remains obscure.

As soon, however, as the formation of the pouch has once begun, there are many influences to make it grow larger. Each successive bit of food, as it glides by, presses upon this yielding and inelastic spot. Gradually a little sac is formed, in which bits of food lodge. These exercise a constant pressure upon the walls of the pouch, and by their weight drag it bodily downward. The larger the pouch the more it holds, and consequently the more it grows. Thus a pressure diverticulum of the smallest size originally may gradually attain a diameter of four inches or more. The general shape of the diverticulum may approach the hemispherical, or it may be more cylindrical or pear-shaped.

It is remarkable that, with very rare exceptions, these pressure diverticula are always situated at the beginning of the œsophagus, or rather between it and the pharynx, and almost invariably affect the posterior wall. The pouch hangs, therefore, in front of the spinal column. It pushes out through the lowest fibers of the inferior constrictor of the pharynx; and the feebleness of this muscle is apparently a potent factor in determining the precise point of origin.

The cases thus far seen have been almost all in men, and at a rather advanced age. A few cases have occurred in children.

Traction diverticula are much more common, but in most instances have little interest except for the pathologist. They are not infrequently found unexpectedly at the autopsy. Rokitansky, and later Zenker, have given explanations of their occurrence: some tissue, which has formed adhesions to the œsophagus, contracts and gradually pulls out the œsophageal wall in the shape of a funnel. Bronchial glands are apt to be the seat of the contractile change. These glands are situated near the bifurcation of the trachea, and accordingly the traction diverticula occur oftenest at this level. There may be two or three in one subject. They are rarely over a third of an inch in depth. From within, the mucous membrane, much wrinkled transversely, is seen to be drawn toward the apex of the diverticulum. The wall of the latter consists either of the mucous membrane alone, bulging out like a hernia, or of the mucous membrane covered by the muscular layer. Inasmuch as children quite often suffer from suppuration and caseation of bronchial glands,

with subsequent shrinkage, we see why traction diverticula are frequent in children.

Clinical History.—The large pressure diverticula always cause grave symptoms, for they obstruct more and more each day the passage of food. At first there is scarcely any disturbance. Gradually, however, deglutition is impeded. A portion of the food lodges in the pouch, and is either wholly or in part regurgitated, though perhaps not immediately. Decomposition is apt to take place in the contents of the diverticulum, giving rise to foulness of the breath and to nausea. The danger reaches its climax when the distended sac presses sidewise upon the œsophagus and closes its lumen, so that no food reaches the stomach. After protracted strangling and vomiting, the material may be in part ejected, and the patient enabled once more to swallow.

Of course the symptoms in individual cases depend upon the mechanical conditions present, and they may vary greatly. Patients contrive all sorts of manipulations, by which they manage to get at least some portion of their food down. Such individuals may maintain a tolerable degree of nutrition for years, although they scarcely ever are in a normal condition. But at last some cause or other renders the amount of food ingested inadequate, whereupon a rapidly progressive marasmus sets in, and the patient will inevitably starve to death unless some relief is afforded.

The most valuable objective evidence in these cases is gained by the use of the œsophageal sound. If the sound enters the sac its passage is impeded. If it happens to slip by the mouth of the diverticulum, it glides readily into the stomach. This varying result may sometimes be obtained at one sitting by repeated trials, and is of the greatest importance in making the diagnosis.

In some instances where the sac was large, a tumor in the neck has been observed at one side of the trachea, appearing after eating and disappearing when the sac emptied itself. Symptoms due to compression of the recurrent and phrenic nerves and of the blood vessels have been noticed in some cases.

Auscultation of the œsophagus during the act of swallowing has been practiced, and of late years œsophagoscopy has been used in the diagnosis of these conditions. Whether these methods of investigation will prove valuable for diagnosticating diverticula, experience must determine.

The traction diverticula are usually of no clinical importance. They do not affect deglutition at all, and their size is too limited to permit any great accumulation of food in them. There is but one way in which they are dangerous: the apex of the funnel may undergo ulceration and perforation. A foreign body, such as some bit of food, produces necrosis of the wall, by what is probably at first a purely mechanical irritation. The tissue ulcerates; and then the inflammation may gradually progress till it causes a severe and often fatal illness. The most frequent event is perforation into a bronchus, followed by the aspiration of food and pulmonary gangrene; or the perforation may take place into the pleural cavity, exciting an ichorous empyema. In other cases the pericardium or a large vein has been perforated. Many a case of apparently spontaneous pulmonary gangrene, or purulent inflammation of the anterior mediastinum, or empyema, has been found at the autopsy to have been brought about in the manner above indicated. These occurrences are fortunately, however, exceptional.

Treatment.—The only possible way of treating the large pressure diverticula successfully is by operation. If surgical treatment cannot be carried out, our efforts are confined to sustaining the patient. If he cannot swallow, we must try to feed him through a tube. As long as this is possible, starvation is averted. It is well to have the patient pass the tube himself. He will find out how best to avoid the sac and reach the stomach. If food can no longer be given in this way, there remain two alternatives: rectal feeding (*vide infra*), or making a gastric fistula. As to the latter, there has been thus far very little practical experience, because cases are so rare.

The traction diverticula admit of no special treatment. If the events above mentioned occur, we must endeavor to meet the indications of the individual case.

CHAPTER III

STENOSIS OF THE ŒSOPHAGUS

Ætiology and Pathology.—Contractions of the œsophagus occur with such relatively great frequency that they are the most important of all its disorders. They originate in various ways. By far the commonest cause is ring-shaped carcinoma of the tube. The new growth in the mucous membrane encroaches more and more upon the lumen of the œsophagus, until finally it fills it. Carcinoma will be discussed at length in the next chapter. We shall here confine our attention to its purely mechanical action in causing stenosis.

Œsophageal tumors other than cancer are very rare. Fibrous pedunculated polypi have been observed a few times. They usually originate in the lowest portion of the anterior wall of the pharynx, hanging down into the œsophagus, which they may thus obstruct. In rare cases sarcoma of the œsophagus has been observed.

A second cause of stenosis is the contraction of cicatrices of the œsophageal wall. The most frequent occasion for this is the extensive ulceration caused by caustic poisons, such as concentrated acids or alkalies. If the victim escapes a speedy death, he is almost certain to have extensive scars formed in the wall of the œsophagus. These scars radiate irregularly in all directions, and, contracting, may almost completely close the tube.

Ulcers from other causes, resulting in stenosis due to the scars they leave, are among the rarities. The possibility of cicatricial œsophageal strictures subsequent to diphtheria has already been referred to (page 496). Further, syphilis has been the well-established cause in some instances, and Quincke has described a few cases in which there were ulcers at the lower end of the œsophagus analogous to the round ulcer of the stomach, or "ulcer due to digestion" (*vide infra*). These ulcers also may eventually produce cicatricial stenosis.

A third and rare cause of stenosis of the œsophagus is compression from tumors external to it. Such swellings may originate in the thyroid gland, or in the lymph-glands of the neck or the anterior mediastinum; or the swelling may be due to a vertebral abscess or an aortic aneurism. This form of stenosis is seldom extreme, for the portion of the tube pressed upon is usually limited.

Next on the list after stenosis due to compression is usually placed what is called intermittent dysphagia (*dysphagia lusoria*). This term is applied to the difficulty in swallowing which is said to be caused by an anomaly in the course of the right subclavian artery. The artery is given off as the last branch from the arch of the aorta, and runs toward the right side just behind or just in front of the œsophagus. It seems, however, *a priori* improbable that the feeble pressure of this vessel as it pulsates should impede deglutition; nor has it yet been proved to do so. It would be more natural to believe, what was indeed the original explanation of the phenomenon, that a large morsel of food passing down the œsophagus compresses the vessel and thus excites uneasiness and palpitation; but so far as we know even this is extremely rare, if it ever occurs.

Stenosis due to foreign bodies belongs to surgery. It need not be said that the clinical symptoms differ greatly in different cases. Not only the obstruction, but also a possible laceration and consequent inflammation are to be considered. Occasionally thrush has been abundant enough to cause pronounced symptoms of stenosis.

Above the point of stenosis, no matter how the condition arose, if only it is well developed and has lasted a certain length of time, the circular fibers of the muscular coat are more or less hypertrophied. This hypertrophy is due to the increased muscular contraction required to propel the ingesta downward. In many cases the tube is also diffusely dilated above the stenosis.

Symptoms.—The effect of every œsophageal stenosis is to render deglutition difficult. If the case is a mild one, the patient experiences nothing more than a moderate pressure in the œsophagus upon swallowing. He feels that the morsel is longer than usual in reaching the stomach. Very soon he notices that solid food and large morsels can be swallowed only with difficulty. Accordingly, he is gradually led to confine himself to a liquid diet, takes small mouthfuls, and always washes down any solid food with a swallow or two of liquid. The narrower the stenosis, the more he is troubled. Finally, even liquids can be taken only slowly and in sips.

It must not be thought that the dysphagia just described is due exclusively to the mechanical obstruction of the lumen. Sometimes a patient is almost entirely unable to take nourishment, and yet at the autopsy no adequate mechanical obstruction is found. The dysphagia must therefore be due to some lesion of the muscular coat of the œsophagus. The impaired contractility of the muscular coat at the affected spot is always a potent factor in impeding deglutition.

As soon as the dysphagia has become considerable there is usually regurgitation of food. At first only a portion of the food comes up, but at last all of it. If the tube has become dilated above the stenosis, food may collect for some hours, and then be regurgitated, mixed with an abundance of very tenacious mucus. We saw a case of this kind in which the patient could fill the sac above the stricture with quite a large amount of fluid without a drop reaching the stomach. If he bent his head sharply forward, the collected fluid would run out again through his mouth. It was not until the pouch was completely filled that a small amount of liquid would trickle through the stenosis into the stomach.

Although the dysphagic symptoms above described generally imply œso-

phageal stenosis, the diagnosis cannot be really established without using a sound. Upon introducing this, it is usually easy to detect the obstacle, which may either allow the instrument to pass with a noticeable jerk, or else prevent its further progress. By measuring the length of the portion introduced before the stenosis is reached we can learn its position. On the average, the entire distance from the teeth to the cardiac sphincter is in adults 16 inches (40 cm.); from the teeth to the beginning of the œsophagus, 6 inches (15 cm.); and consequently the length of the latter is about 10 inches (25 cm.). If we succeed in passing a smaller sound through the stricture, the feeling as we move it back and forth will give us some idea of the length of the stenosis, or will demonstrate the existence of several lying one below the other, etc. If the end of the sound can be moved about very freely above the stenosis, we may conclude that the tube is dilated there.

Hamburger has employed auscultation of the œsophagus for diagnostic purposes. If we listen behind, to the left of the upper dorsal vertebræ during deglutition, we hear a gurgling sound, due to the act of swallowing, extending down the tube to the stenosis, but no farther. Then come all sorts of sounds, some of them caused by the fluid trickling slowly through the narrow part, and some caused by regurgitation. In general, the results obtained by auscultation are rather variable and uncertain, and it is therefore little used in practice.

Œsophagoscopy, though technically difficult of application, has nevertheless been frequently very successful. In many cases the X-ray examination of the œsophagus, filled with a mixture of bismuth and mashed potato, gives very clear pictures. The retention of the pasty mass above the stenosis is distinctly seen. Fluoroscopy after the swallowing of a "bismuth pill" (a gelatin capsule filled with the subnitrate of bismuth) also gives important information. It is possible closely to follow on the fluoroscopic screen the descent of the pill, its arrest, etc.

Having established the fact of the existence of a stenosis, we have next to determine its nature, which is our chief guide to prognosis and treatment. In certain instances the history of the case gives us the needed information. The diagnosis of cicatricial stricture can hardly be made unless the patient himself tells us of being burned or injured by caustic poisons. The previous history is likewise of great importance if the stenosis be due to foreign bodies, to diphtheria, or to syphilis. If no decisive ætiological factor can be elicited, we must carefully examine the neck and thorax, with regard to the possible existence of a swelling compressing the œsophagus. When an aortic aneurism has acted in this way, a rhythmical movement has sometimes been communicated to the free end of a sound introduced as far as the stenosis. If the physical examination does not reveal a compressing tumor, and particularly if the stenosis has developed gradually and in an elderly person, we are almost compelled to assume that there is cancer of the œsophagus. This is, after all, by far the most frequent cause of œsophageal stricture. If the new growth has ulcerated, a little portion of it may adhere to the end of the probe, and, on microscopic examination, render our diagnosis of carcinoma certain. Œsophagoscopy is also a means of making a certain diagnosis.

The prevailing characteristic in stenosis of the œsophagus is inanition, increasing as the dysphagia increases. The patient gets to be very much

emaciated, and so feeble that he cannot leave his bed. The temperature is subnormal; for weeks it keeps at 95° to 97° F. (35° to 36° C.). The pulse grows very small and slow, being 40 to 60 per minute. The heart sounds are soft. Respiration is superficial and slow; and toward the close of life short pauses occur after expiration, before inspiration begins. The stomach and intestines are so empty that the abdomen is very concave, while the abdominal walls usually feel tense and resistant. In all cases when the nature of the stenosis precludes the possibility of cure or improvement, death results from increasing exhaustion, the lamp of life gradually flickering out.

Prognosis and Treatment.—In prognosis the main factor is, of course, the nature of the stenosis. If it is due to foreign bodies or to cicatrices, it may be completely cured. In stenosis from other causes it is often possible to produce considerable improvement, at least temporarily. The final result must be confessed to be usually unfavorable, as we should expect from the nature of the original trouble.

The treatment is chiefly mechanical. We shall not speak of operations for the removal of new growths, etc. What we do refer to is a methodical and gradual dilatation of the stricture. Its results are sometimes brilliant, particularly in cicatricial stenosis. Other varieties, such as the stenosis from cancer, may sometimes undergo considerable though but temporary improvement with this treatment.

The best instrument to employ is the flexible, so-called English, œsophageal bougie. It is made in all sizes. If the stenosis is very narrow indeed, we may have to resort to catgut at first. Whalebone bougies, with olive-shaped ivory tips of various sizes to screw on the end, are also good, except that, being stiffer, there is more danger in using them. For introduction of the bougie, the patient should be seated, with the head slightly extended backward. The first two fingers of the left hand are introduced into the throat and guide the instrument, previously well oiled, over the back of the tongue and the epiglottis into the œsophagus. Of course, no violence must be used. Otherwise a perforation might occur if there were a soft, broken-down cancer, or an aortic aneurism. However, such a misfortune is very exceptional.

The use of the bougie is almost invariably beneficial if the stricture can be passed. The patient generally finds that he can swallow easier than before, and will himself request a repetition of the performance. If the patient is an intelligent person, it is advisable to have him introduce the bougie himself. Patients often acquire even greater skill with it than the physician has. The bougie should be passed regularly once a day, or, at most, twice daily; and in favorable cases we shall be able gradually to increase the size. If so, the symptoms speedily abate, and, with the increased ingestion of food, the patient gains flesh very fast.

If the stenosis is extreme, and, although it admits the bougie, does not allow of sufficient nourishment, we must pass a tube into the stomach through which to introduce liquid food. Milk is the best food to choose. Raw eggs, sugar, wine, etc., may be mixed with it. The various infants' foods and the artificial proteid preparations are also excellent. Their consistence is favorable for the purpose, and they supply a considerable amount of nourishment in a small bulk. Until recently the sounds and dilators employed in stenosis of the œsophagus have been usually introduced by the mouth, but of late

the practice has been constantly growing of treating stenosis through an artificial opening in the œsophagus or stomach. Mechanical dilatation can thus be accomplished with much greater rapidity and completeness. In the surgical wards in Erlangen admirable results have been obtained by using sounds after œsophagotomy in cicatricial stenosis; whereas in carcinoma of the œsophagus the improvement is, of course, merely temporary, although the symptoms are greatly alleviated. Further particulars with regard to the surgical treatment of œsophageal stenosis are to be sought in text-books on surgery. [See also remarks on page 339.]

Thiosinamin may be considered in the treatment of the cicatricial stenoses of the œsophagus. A fifteen-per-cent alcoholic solution is used, and about 1 gr. (gm. 0.05) of thiosinamin is injected every other day, subcutaneously, into the front of the chest. The cicatrices are supposed to become so dilatable that the introduction of even large bougies is easy, and thus a complete recovery may be brought about. Considering the experience to date, further trials of this interesting remedy are certainly indicated.

Finally, we desire to append a few remarks as to the nourishment of patients by enemata, to which we are particularly apt to have recourse in cases of œsophageal disease.

Brilliant results are never to be expected from rectal feeding. It is indeed probable that life may be by this means somewhat prolonged, but not indefinitely. The moral effect, however, is very valuable, when the patient could otherwise receive no nourishment whatever. The sufferer feels that something is being done to avert absolute starvation. The simplest materials for the nutrient enemata are milk, solutions of grape sugar, eggs, albuminous preparations, and the like; to which we may add pepsin and pancreatin in the hope of promoting absorption. Before every nutrient enema, the rectum should be cleansed by an enema of plain water. Too large an amount ought not to be given at one time, say about 2 oz. (gm. 50), and the fluid must be raised to about the temperature of the body. [The limit in quantity set by the author is smaller than many patients can retain. If the fluid is injected gently and slowly, 4 to 8 oz. may be administered every six hours, and cases are on record of patients retaining even 16 oz. The rectum seems capable of a certain amount of training in this regard, so that the amount given may eventually be greater than at first. Unusual irritability may be diminished by adding to the enema about 5 minims of laudanum.]

CHAPTER IV

CANCER OF THE ŒSOPHAGUS

Ætiology and Pathology.—Cancer is the most important and most frequent affection of the œsophagus. We have already mentioned in the preceding chapter that often stenosis is the result of carcinoma in the œsophageal walls.

Little is known about the ætiology. It has been often maintained that mechanical, chemical, or thermic irritation of the mucous membrane may result in the development of cancer; but this is not certain. It receives some support from the remarkably frequent occurrence of œsophageal cancer in hard drinkers. Now and then the patient himself will allege a perfectly

definite cause for his disease, such as the lodging of a foreign body, or the swallowing of a very large or very hot morsel. Still, it is hardly possible in any particular case to decide how much value such statements have. Carcinoma may sometimes develop in the scar of an old ulcer. This is of interest when we recall the similar fact in regard to gastric carcinoma (*vide infra*).

Œsophageal cancer follows the general rule in being most frequent in elderly people—somewhere between forty and sixty years of age. The male sex is decidedly more often attacked than the female.

As we might expect from the histological character of the epithelium lining the œsophagus, primary cancer here is invariably composed of pavement cells. The new growth may be either hard, firm, and fibrous, or it may be soft, succulent, and but scantily supplied with connective tissue. The first variety corresponds to the "scirrhus" of older writers, and the second to "medullary" cancer. Usually the new formation encircles the entire tube like a ring, extending three to ten centimeters longitudinally. Exceptionally a still larger portion of the œsophagus is involved, sometimes almost all the mucous membrane. The tumor is usually seated in the lower and middle thirds of the œsophagus, being much rarer above.

Symptoms and Complications.—In the great majority of cases the symptoms are those of a gradually increasing stenosis, with its results. We may therefore refer to the preceding chapter for most of the particulars. There are, however, exceptional cases in which the carcinoma is flat and entails no dysphagia, or so little that œsophageal trouble may not be suspected. We have repeatedly seen cases of extensive secondary hepatic cancer, or of pulmonary gangrene (*vide infra*), in which the real primary disease was a flat cancer of the œsophagus, which gave no clinical signs of its existence, and was therefore not diagnosticated.

It is characteristic of the stenotic symptoms produced by œsophageal cancer that sometimes a considerable and apparently spontaneous amelioration occurs. This is because of a superficial ulceration of the new growth. The tumor is transformed into an ulcer, and one can easily understand how this may occasion a temporary improvement in deglutition. Not infrequently, small hemorrhages may occur from these ulcerations.

Important clinical symptoms may result from conditions secondary to the new growth. The cancer may extend to neighboring organs. Not infrequently the cardiac extremity of the stomach is thus involved. Sometimes such a tumor may be felt in the epigastrium; but in most cases there is nothing to indicate that the stomach is attacked.

The neighboring parts of the trachea or bronchi are sometimes affected, and important symptoms result from such a complication. If perforation occurs, there is a pronounced irritative cough almost every time the patient takes food, and an almost certain result is the inhalation of food or of decaying bits of the tumor, with consequent pulmonary gangrene, and, as a rule, speedy death. Perforation of the pleura with putrid pleuritis has also been observed. The pericardium and the aorta may likewise be attacked. A few instances are known in which the vertebræ have been involved, the spinal cord compressed, and paraplegia thus induced. We have ourselves seen one such case.

Quite frequently the left recurrent nerve is affected, and a paralysis of the vocal cords is produced, which can be detected by the laryngoscope. This

nerve lies so close to the œsophagus that it is peculiarly exposed to injury from the new growth itself, or from any inflammatory process which may be set up around it.

Metastatic cancer in distant organs is not infrequent. It attacks most often the liver; the lungs, kidneys, pancreas, bones, and brain are also liable to it.

Pulmonary gangrene must be mentioned as a relatively frequent complication, and one which has serious consequences. We have already stated that it may result from perforation. Even without actual perforation, an infection of the air-passages may take place. A still more frequent cause of pulmonary gangrene is the inspiration of decaying masses vomited or regurgitated.

Clinical History, Termination, Prognosis, and Treatment.—The disease is incurable. Operative removal has never been successful, notwithstanding repeated attempts in this direction. [Recently attempts at radical operation for malignant growths situated in the lower and more accessible portion of the œsophagus have been made by Willy Meyer, of New York, and others, with the employment of an air-pressure apparatus. Although, thus far, the results have not been successful, the prospect for a radical cure of an early case is very promising.] The entire duration of the disease seldom exceeds a year, or a year and a half. At the end of this period the patient dies either from lack of nourishment or as a result of some one of the complications above enumerated. Treatment is purely symptomatic. Temporary improvement may be obtained by mechanical treatment of the stenosis. A gastrostomy is indicated when the patient is no longer able to take nourishment by mouth.

CHAPTER V

RUPTURE OF THE ŒSOPHAGUS

Medical literature records a small number of cases which prove that the sudden rupture of the œsophagus in persons previously perfectly well is possible, although, of course, very rare. The first and most famous instance was described by Boerhaave in 1724.

The symptoms, according to the observations thus far reported, usually commence with sudden nausea and vomiting, during or shortly after a hearty meal. There is simultaneously an extreme, general collapse. There is pallor of the face and extremities, cold perspiration, and an extremely feeble pulse. Sometimes the patient feels a sudden darting pain in the chest. Almost invariably an extensive emphysema overspreads the neck and thorax. Death results in a few hours, or at latest in a few days.

The autopsy reveals a tear in the œsophagus, invariably situated in its lower half. It may be five centimeters long, and it is almost always longitudinal. Food has usually escaped into the surrounding tissues, in which case a secondary purulent inflammation exists, if death was not immediate.

Zenker has attempted to explain this remarkable phenomenon by a supposition that œsophagomalacia always precedes these so-called spontaneous ruptures. The cause of this softening of the œsophageal walls is perhaps the action of gastric juice escaping into the tube and attacking a surface which, through some temporary disturbance in the circulation, has lost its normal powers of resistance.

CHAPTER VI

NEUROSES OF THE ŒSOPHÀGUS

1. **Spasm of the Œsophagus.**—In rare instances œsophageal disturbances are observed, which appear to result from spasmodic contraction of its muscular coat. Nervous and hysterical subjects are particularly apt to present temporarily the symptoms of extreme stenosis, for which there is no anatomical basis. Such cases are termed "spastic stenosis" of the œsophagus, or "œsophagismus." It is, of course, possible that there may exceptionally be some real lesion at the foundation of the trouble, and that the spasm is the reflex result of an ulcer or inflammation affecting the œsophagus. It is even affirmed that the reflex influence may sometimes originate in distant organs, such as the uterus; but the exact nature of these reflex spasms is at present exceedingly obscure (see chapter on Hysteria). The dysphagia is usually attended by a painful sense of constriction in the throat and chest. The bougie comes upon an obstruction, which usually soon yields. This circumstance, that when the spasm relaxes it is possible to introduce the bougie without any difficulty, confirms the diagnosis. Other important factors are, the character of the symptoms as a whole and the other attendant nervous and hysterical disturbances. Some authors also explain the "*globus hystericus*"—that feeling as if a lump were passing up or down in the throat and chest—as a spasm of the œsophagus.

2. **Paralysis of the Œsophagus.**—Of this subject we have little accurate knowledge. It is not improbable that an extensive bulbar paralysis, affecting the muscles of the pharynx and larynx, may sometimes involve the œsophagus; although such a disturbance hardly ever gives rise to prominent symptoms in this disease. Ziemssen asserts that sometimes the œsophagus seems to participate in post-diphtheritic paralysis when extensive.

SECTION IV

Diseases of the Stomach

CHAPTER I

BRIEF PRELIMINARY REMARKS ON THE EXAMINATION OF THE GASTRIC CONTENTS[1]

When a disease of the stomach exists, we have the subjective symptoms to consider, such as anorexia, eructations, vomiting, and gastric pain, together

[1] Complete particulars with regard to the methods of examining the gastric contents cannot be given here, but they may be found in the following works: Ewald, "Klinik der Verdauungskrankheiten"; Boas, "Diagnostik und Therapie der Magenkrankheiten"; Leo, "Diagnostik der Krankheiten der Verdauungsorgane"; Riegel, "Erkrankungen des Magens, in Nothnagel's Handbuch," etc.

with the external physical signs obtained by inspection and palpation, such as tenderness, distention, peristaltic motion, swelling, and tumors; and we also have for our guidance an examination of the stomach and the stomach contents. The procedure was introduced by Leube, followed by Ewald, Boas, Riegel, and many others. It is carried out by means of the stomach tube, and is now universally employed among physicians, for in fact its results are so important and decisive with regard to diagnosis, that an examination of this sort is at the present day indispensable in any severe case of persistent gastric trouble. The inconvenience of the investigation for the patient is comparatively slight, especially now that a soft and yielding tube (Nélaton), or the so-called Jacques' patent rubber tube, is employed. This should have free apertures at its lower extremity, and it is introduced into the stomach in the following manner: The tube is moistened with water, put into the patient's mouth, and passed backward over the base of the tongue, and he is told to swallow it, as it were; the physician at the same time aids by pushing the tube, but he does not introduce his finger into the patient's mouth. In this way it is almost always easy to introduce the instrument into the œsophagus, and so into the stomach. Most patients, if the tube has to be employed frequently, soon learn to guide it themselves. Formerly a wire was placed inside the tube so as to stiffen it for introduction, but this is ordinarily useless, if not a hindrance.

If the tube has been introduced into the stomach, and we wish to obtain a portion of the contents of that organ for examination, it can, in most cases, be got by simple "expression." If the patient makes his abdominal muscles tense ("presses"), or if he makes a few slight efforts to vomit, there is usually a sufficient amount of the gastric contents expressed through the tube into a beaker held to receive it. This portion of the gastric contents is filtered, and the filtrate subjected to examination. If we wish to empty the stomach completely ("rinse it out"), the upper end of the inserted tube is connected, by means of a short piece of glass tubing with a longer rubber tube connected at its other end with a large glass funnel which holds about a liter of water. If warm water is poured into the funnel, and this is alternately raised and lowered, we shall eventually entirely empty the stomach (see Fig. 70). It is advisable to insert a short bit of glass tubing in the rubber tube (fenestra), so as to observe whether the fluid runs well in either direction. Kussmaul originally employed a "stomach pump" for rinsing out the stomach, but this has been quite universally abandoned for the simple siphon apparatus suggested by Hegar.

By using the stomach tube we are readily enabled to settle the following important points: First, the chemical and other constituents of the gastric juice, or contents (the secretory activity of the stomach); second, the motor activity of the stomach; and third, the size and position of the organ.

1. **General Nature of Gastric Contents.**—Before we attempt a more exact chemical analysis of the gastric contents, we should first make a general inspection of the entire amount which has been obtained by the stomach tube. The macroscopic differences in the subdivision of the food are in themselves very striking. If the total gastric contents is changed into a finely granular mass, this, as a rule, points to a satisfactory peptic quality of the gastric juice and a normal HCl content. In anacidity of the gastric juice, the food is

found in large pieces, just as it has been swallowed. In such cases, the opening of the tube is more easily occluded than it is, for example, in cases of hypersecretion, where the entire gastric contents are changed into a fine pap. The demonstration of the presence of profuse, stringy mucus in the gastric contents is likewise of importance. This can be easily recognized by pouring the contents from one vessel into another. If there has been gaseous fermen-

FIG. 70.—Method of washing out the stomach. (Erlangen Medical Clinic.)

tation, the gastric contents present a foamy appearance. On standing, such gastric contents separate into three layers: an upper layer of foam, a middle of fluid, and a bottom layer of more solid particles. We must look very carefully for the possible presence of blood (*vide infra*). The odor of the gastric contents is also important. Fatty acids are usually readily recognized by their rancid odor. Also in putrid decomposition, which occurs frequently in carcinoma, the odor enables us to recognize the condition. In gaseous

fermentation there is a slightly pungent odor of carbon dioxid and aromatic substances.

We shall add here a few remarks on the microscopic examination of the gastric contents. This enables us, first of all, to recognize food remnants. With profuse HCl secretion the meat is well digested, but there are still many starch granules present. On the other hand, with anacidity the digestion of albumen suffers most. We often find fermentation produces yeast molds, long-extended bacilli of lactic-acid fermentation, or sarcinæ (*vide infra*). Finally, we occasionally see red blood corpuscles, leucocytes, epithelial cells, or tumor particles. The diagnostic significance of all these findings is obvious.

2. **Free Hydrochloric Acid, Pepsin, and Lactic Acid. Constituents of the Gastric Juice.**—In a healthy person the stomach is almost completely empty in the morning before breakfast—i. e., some ten or twelve hours after the preceding evening meal. If we introduce the stomach tube we obtain, therefore, by expression either nothing at all or a very small amount of watery mucus, of neutral reaction. If this fluid contains a little hydrochloric acid, this is not necessarily anything abnormal, but if there is a large amount of acid fluid in the fasting state, disease is indicated. The healthy stomach contains in the morning few vestiges of the food swallowed the day before. If we pour in some water, this is returned almost perfectly clear. If now the subject of examination eats a so-called test breakfast, consisting of an ordinary roll and a large cup of weak tea, at once the stomach begins to secrete its peculiar juice. If the stomach tube is introduced an hour after a test breakfast has been taken, and a sufficient amount of the stomach contents is expressed and filtered, we shall obtain a fluid of acid reaction, and, under normal conditions, invariably containing free hydrochloric acid uncombined with albumen. It is the comparatively small amount of the test breakfast which makes it most suitable for the determination of the important question, whether the stomach secretes hydrochloric acid in a satisfactory manner. If we introduce into the stomach a larger amount of food ("test meal," *vide infra*) there will, indeed, in most cases, be free hydrochloric acid after one or two hours, but the amount of albuminous material present is so great that even under normal circumstances all the hydrochloric acid secreted is taken up by the albuminous substances, and consequently there is no hydrochloric acid to be discovered by our ordinary chemical tests. If, on the other hand, we do not find any free hydrochloric acid in the gastric contents, or, at any rate, merely doubtful traces of it, an hour after the test breakfast, this condition is termed anacidity, or subacidity, and it must be regarded as abnormal, particularly if repeated examination leads to the same result. The term acidity in this connection relates to the hydrochloric acid. Even if this is absent it is perfectly possible that there may be other organic acids in the gastric contents.

The test for the presence of free hydrochloric acid now almost universally employed is one introduced by Günzburg, who recommended a solution of 2 parts of phloroglucin and 1 part of vanillin in 30 parts of absolute alcohol. If we mix a few drops of this phloroglucin-vanillin in a saucer with a few drops of the filtered gastric contents to be examined, and then heat the mixture cautiously so as to avoid charring, there will immediately develop on the

edge of the fluid a beautiful red border, if free hydrochloric acid is present. Another good reagent for free hydrochloric acid is methyl violet. This requires an accurate comparison of colors, so that the weak watery solution of methyl violet is divided into two portions in test tubes. If now we add to one test tube some of the gastric contents containing free hydrochloric acid, the violet is immediately changed into a distinct blue color, which is evidently different from the original fluid contained in the other test tube. For a third test what is called Congo paper is employed—i. e., strips of paper colored with an aqueous solution of Congo red. If the stomach contents contain free hydrochloric acid, the red color of the paper is changed to a distinct blue. There is a certain inaccuracy in this last-mentioned test with Congo red, as the red is colored blue by concentrated solutions of other acids, particularly lactic acid; but, as a matter of fact, the other acids are scarcely ever present in the stomach in sufficient amounts to invalidate the test; so that in actual practice, if the Congo paper changes to a distinct blue, we may almost always infer that free hydrochloric acid is present. Still, it is advisable in every case to carry out the two other tests also, which are almost as simple and entirely unambiguous. There is a reagent often employed which indicates the presence of free acids in general, including free lactic acid. It consists of an alcoholic (or aqueous) solution of "tropæolin OO." The yellow color of tropæolin is changed to a beautiful red by even small amounts of acid, but still more readily by hydrochloric acid than by the organic acids. Blue litmus paper, as is well known, is colored red by free acids as well as by acid salts.

Sahli has suggested an ingenious method to demonstrate the presence of free hydrochloric acid. He prepared small bags filled with methylene blue and tied with a catgut thread—so-called desmoid capsules. If the patient swallows such a capsule after a meal, then, if free hydrochloric acid is present, the connective tissue of the catgut is digested, the methylene blue is set free in the stomach, and after about two hours the urine is colored a distinct greenish blue. In exceptional cases the blue discoloration occurs only after the addition to the urine of acetic acid. If the stomach contents do not contain free hydrochloric acid, the catgut will remain undigested, the capsule enters the intestine unopened, and is expelled without the urine becoming blue. Because of its great simplicity, this test can very well be used in practice, although, on account of all kinds of accidents, there are decidedly more sources of error to be feared than in the direct examination of the siphoned gastric contents. In the Breslau clinic we used the preferable keratin capsule instead of the desmoid capsule of Sahli.

If the examination of the contents of the stomach an hour after the test breakfast shows the presence of free hydrochloric acid, an experienced observer will often be able to estimate from the degree of the qualitative reaction whether he is dealing with a small or considerable amount of hydrochloric acid. An accurate quantitative estimation is possible by titration of the stomach contents with a decinormal soda solution, for which a few drops of phenol-phthalein solution serve as index.[1] The amount of acid determined by this process is, of course, the sum total of free acids, and not exclusively

[1] We scarcely need to describe this simple process more fully.

the free hydrochloric acid. Still, provided there is an abundant amount of hydrochloric acid in the gastric contents, the results obtained may be ascribed without great error to hydrochloric acid alone; for experience has shown that, when there is a considerable amount of hydrochloric acid in the gastric contents there is scarcely ever any considerable amount of organic acids (*vide infra*). The degree of acidity of the gastric fluid is usually expressed in the number of cubic centimeters of soda solution which are necessary, as shown by the titration, to neutralize 100 c.c. of the gastric fluid; for example, if we have required 6 c.c. of soda solution to neutralize 10 c.c. of the gastric contents, the acidity is reckoned at 60. In terms of hydrochloric acid we would have 60 multiplied by 3.65, equal to 19 mgm. of hydrochloric acid—that is, 0.22 per cent. The degree of acidity which is found in the gastric contents of healthy persons after the test breakfast is usually 55 to 65; amounts of 70 to 80 indicate hyperacidity. If the gastric juice contains an abundance or an excess of hydrochloric acid, the further question, and an important one, arises whether there is a hypersecretion, or, more correctly, a continuous secretion of gastric juice. As we have already mentioned, the normal stomach does not ordinarily secrete except when its mucous membrane is affected by food swallowed, or by other stimulating influences. The fasting stomach does not, as a rule, contain any hydrochloric acid, and when the stomach has passed the food on into the duodenum, its secretion immediately ceases. In order, then, to determine the existence of hypersecretion or continuous secretion, we must examine the contents of the fasting stomach at last six or seven hours after the last meal. If we then find fluid in the stomach, with distinct or abundant amounts of hydrochloric acid contained in it, we are justified in assuming that there is an abnormal hypersecretion on the part of the stomach.

Physiology teaches us that not only hydrochloric acid, but pepsin in association with it, is necessary for the peptonizing of albuminoids. The demonstration of pepsin in the gastric contents is, therefore, essential to a determination of the peptic function of the stomach. Experience has shown, however, that pepsin is rarely absent from the gastric juice, and if there is a distinct secretion of hydrochloric acid we may almost always assume that pepsin is secreted, without making any special examination. Even in cases of anacidity pepsin often continues to be secreted. Still, there are cases when the direct demonstration of the presence or absence of pepsin in the gastric juice is desirable. The process is simple. We obtain some gastric contents in the ordinary way after a test breakfast, filter it into a test tube, and put into it a square shaving of the white of a hard-boiled egg. Normal gastric juice containing hydrochloric acid and pepsin, if kept at a temperature of 98.6° F. (37° C.) in an incubator, will dissolve the flake of albumen completely in from thirty to sixty minutes. If the gastric juice contains no hydrochloric acid, but only pepsin, the albumen will be dissolved if we add a few drops of dilute hydrochloric acid. If, however, pepsin also is absent from the gastric juice, the bit of albumen will swell up but will not be actually dissolved, unless we add artificial pepsin.

Besides hydrochloric acid there are also some organic acids in the gastric juice; the most important of these is lactic acid. The lactic acid is not a product of the gastric mucous membrane, but the result of lactic-acid fer-

mentation of the carbohydrates in the gastric contents, except for small amounts which may be directly introduced with the food. If we test the gastric contents a short time (fifteen to thirty minutes) after a test breakfast, we can often find some lactic acid present, but as yet no hydrochloric acid. This corresponds to the amylolytic stage of gastric digestion. Later, when the secretion of hydrochloric acid has begun, the lactic-acid fermentation promptly ceases, so that under normal conditions, as we have said, we find hydrochloric acid distinctly present an hour after the test breakfast, but usually no lactic acid. If we do find lactic acid it is always an indication of an impaired production or absence of hydrochloric acid. There is a condition which is especially favorable for the formation and collection of large amounts of lactic acid in the stomach. This is when the absence of hydrochloric acid is associated with a stagnation of the ingesta in the stomach. This combination is especially frequent in the case of carcinoma of the pylorus (*q. v.*). Under these conditions the lactic-acid fermentation may go on unchecked, and we find an abundance of that acid in the stomach at whatever time we make a test.

The qualitative test for lactic acid is by means of Uffelmann's reagent: by adding a drop of ferric-chlorid solution to a three- or four-per-cent solution of carbolic acid, we obtain a liquid of a beautiful steel-blue color. Upon adding to a small portion of this blue fluid, contained in a test tube, gastric contents in which there is lactic acid, the blue color changes to a distinct yellow, or yellowish-green. The test should not be regarded as positive unless there is a distinct yellow color. We may first agitate the gastric fluid with ether, and then make the test with the ether extract, as ether takes up the lactic acid. The ether extract is carefully evaporated, the residue is dissolved in water, and the watery solution is tested with Uffelmann's reagent. The quantitative estimation of lactic acid is tedious, and not very important in practice.

The fatty acids and acetic acid are not found in the gastric contents unless there is marked fermentation, and their presence therefore indicates a stagnation of the gastric contents (*vide infra*), and the absence of hydrochloric acid, which acts as an antiseptic. In practice we form an opinion as to the presence of these acids by the sense of smell, since their demonstration by direct chemical means is far from simple. The presence of the volatile fatty acids is recognized by heating the gastric contents in a test tube, and at the same time exposing a piece of moistened blue litmus paper to the vapors, which turn it red.

3. **Determination of the Motor Activity of the Stomach.**—The employment of the stomach tube also enables us to form with ease a satisfactory estimate of the motor function of the stomach, and this is an extremely important matter. As physiology teaches us, the pylorus remains firmly closed during the first part of gastric digestion. It does not open until the food is sufficiently prepared, and then the chyme is discharged by successive jets into the duodenum. For practical purposes it is sufficient to know that the examination of healthy persons has shown the stomach to be completely empty again about two hours after the ingestion of a small amount of food—e. g., after the test breakfast (*vide supra*). If, then, we rinse out the stomach two hours after a test breakfast we ought not, under ordinary circumstances, to find any

large amount of bread in the wash water. But it is a comparatively easy task for the stomach to make away with the test breakfast; so it is more suitable, if we desire to test its motor powers, to administer what is called a *test meal,* consisting of a plate of broth, 150 gm. (5 oz.) of underdone broiled steak (minced) 50 gm. (2 oz.) of potato *purée,* and a roll. A meal of this sort disappears from the stomach often in three or four hours, but at the latest, under normal conditions, in seven hours. If we wash out the stomach seven hours after a test meal and still find any considerable amount of food, it is sure proof of an unsatisfactory discharge of the stomach contents, whether as a result of impaired motor power or, as is most frequently the case, of a mechanical stenosis of the pylorus. If we really wish to know how long portions may be retained in the stomach, we may add to the meal a few cranberries or green-colored beans, and the like. Such easily recognizable things may sometimes be found again in rinsing out the stomach several days after their ingestion. As a rule, in practice, it is advisable to perform the first gastric lavage in the early morning before the patient has taken food. If the stomach still contains distinct remnants of food taken the previous evening, this in itself is ample proof of a marked retention of the gastric contents.

In this connection, however, we may make brief mention of another method originated by Ewald, with this same object of determining the motor efficiency of the stomach. The patient is given, early in the morning, a gelatin capsule containing 15 gr. (gm. 1) of salol. Salol is not broken up until it reaches the intestine, when it separates into carbolic and salicylic acids. The salicylic acid is then immediately excreted in the urine, and readily detected by chlorid of iron. If the stomach does not empty itself in a normal manner, the salicylic reaction is often to be detected in the urine even after twenty-four or thirty hours, at which time it would have vanished under normal conditions. This test has not been adopted very generally in practice, because it is uncertain.

4. **Estimation of the Size and Position of the Stomach.**—Under normal conditions, the stomach lies mainly in the left hypochondrium, directly below the vault of the diaphragm, and nearly vertical. The pylorus lies in the epigastrium, in the median line or a trifle to the left. There are many individual variations from this position, such as displacement from pressure of the clothes, etc. Many of these changes of position have really no great pathological significance. Still, it is important to determine these points about the organ. An experienced eye not infrequently recognizes the position of the stomach by mere observation of the abdomen, but often this is negative or misleading. By percussion we may distinguish the limits of the deep tympanitic resonance of the stomach from the usually higher tympanitic resonance of the surrounding intestines; or we may mark the lower limit of dullness when the organ is full, so that we can sometimes form a tolerably certain opinion as to the lower border of the stomach. In general, these results are also deceptive and uncertain.

With the aid of the stomach tube the matter is very simple. It is accomplished by blowing up the stomach with air, in the manner first proposed and practiced by Runeberg. This is done by means of the ordinary rubber double balloon bulb [such as is employed with the Paquelin cautery; or with a simple Davidson syringe]. The method used by us in our wards almost every day is as

follows: The patient is either fasting or has had his stomach thoroughly rinsed out, and lies as nearly horizontal as possible upon his back. The stomach tube, being introduced, is connected with the bulb at its upper end, and air is pumped in. As the pylorus is almost always firmly closed, the contours of the stomach become visible very shortly. By percussion of the inflated stomach, we can verify the results of simple inspection.

We should then not only mark the position of the greater curvature but also that of the lesser curvature, if visible. If the stomach is normal in position and size, the protuberance occupies the epigastrium above the navel. If the stomach is dilated, the greater curvature reaches below the navel; while if the stomach as a whole is displaced downward (*vide infra,* gastroptosis), the smaller curvature also falls to the neighborhood of the navel or below it.

As soon as we disconnect the bulb the air escapes, and the distended stomach collapses. We can, if a certain degree of caution is used, repeat the procedure more than once, so as to make sure of our results. Of course, inflation should be interrupted immediately if the distention of the stomach becomes painful; and we should abstain from the procedure altogether whenever there is suspicion of an ulcer, because the artificial distention of the stomach in this case might do harm. In some cases, direct inflation does not render the contours of the stomach distinctly prominent, probably because of insufficiency of the pylorus. In such cases, and in such only, we should employ a rubber bag (condom) fastened on the lower end of the tube, having previously tested its distensibility. When this is introduced into the empty stomach and blown up, we can usually discern it through the abdominal walls and determine the position, but of course not the size, of the stomach. For ordinary cases the first-described method is not only simpler but more satisfactory, and, so far as the patient is concerned, less disagreeable. If the abdominal walls are thick with fat the method fails, but such a condition is very rare when there is severe gastric disease.

The simple method just described seems to us to have rendered entirely superfluous most of the other more or less complicated procedures for the determination of the size and position of the stomach, especially as no decidedly better results are obtained by them. This last statement applies especially to electric transillumination of the stomach and anterior abdominal walls by means of an incandescent light introduced with a tube. In practice the X-ray examination of the stomach filled with a bismuth-potato pap can usually be dispensed with, although this method has given very good results (Rieder, Holzknecht, and others).

In ordinary practice where the introduction of the stomach tube is not always practicable, the old method recommended by Frerich may be employed. For carrying this out it is best to have the patient fasting, and in a horizontal position. Seventy-five to one hundred and fifty grains (gm. 5 to 10) of tartaric acid are given, and immediately after an equal amount of bicarbonate of soda, each drug being dissolved in half a glass of water. At once there is an abundant production of carbonic dioxid. The stomach is blown up and its outlines become sometimes very distinct, both for observation and for percussion. The great advantage of the previously described method of pumping in air consists, however, in the fact that we have the degree of

distention under far better control, and that we can repeat the process several times in succession.

5. **Testing the Absorptive Powers of the Stomach.**—Brief mention should be made of Penzoldt's method of determining the absorptive powers of the stomach. A gelatin capsule filled with iodid of potassium, if swallowed by a healthy person fasting, will give rise at the end of ten or fifteen minutes to a reaction for iodin in the saliva and in the urine (by adding sulphuric acid and shaking with sulphid of carbon). In severe diseases of the stomach the time required is often much longer. In practice, however, the method has not gained any great importance, particularly since the examinations of Mehring have shown that the absorptive powers of the stomach are after all slight. Water is not at all absorbed from the stomach; sugar, peptone, and salt are absorbed to a slight degree, but with a simultaneous excretion of watery secretion. Alcohol, on the other hand, is absorbed in large amounts from the stomach.

6. **Demonstration of Blood in the Gastric Contents.**—Any admixture of blood with the contents of the stomach is of great importance in the diagnosis of certain gastric diseases, particularly ulcer and carcinoma. If there is fresh blood mixed with the vomitus or with the gastric contents obtained by washing out the stomach, it is often directly recognizable from its characteristic appearance, but if the blood is already decomposed or intimately mixed with the food, or present in very small amounts, we need special methods for its certain recognition. Microscopic examination alone is insufficient, for the red corpuscles are soon destroyed in the stomach. The method of spectrum analysis requires a special spectroscope. We search for the characteristic bands of hematin in an ethereal extract, made by shaking up a portion of the gastric contents, to which a few drops of glacial acetic acid have been added, with ether. For medical practice the most useful and simple method is Van Deen's test, with fresh tincture of guaiac and turpentine. A small amount of a mixture of equal portions of these two liquids is poured into a test tube upon a portion of the gastric contents. If blood is present, there appears at once, or after a short time, an intense blue color at the junction of the two fluids. As certain other matters contained in the food may cause this same color reaction, it is more satisfactory first to decompose the gastric contents with a few drops of glacial acetic acid, then extract with ether and perform the guaiac-turpentine test with the ether extract. This test is especially distinct if there are organic acids present, as in case of carcinoma, in which disease the stomach contains blood in association with lactic acid, but no hydrochloric acid. On the other hand, in our experience, the test is often uncertain in the case of ulcer, because the presence of hydrochloric acid is a hindrance. Another common method is to see if we can produce Teichmann's hemin crystals from the gastric contents. The test is not always successful, but if successful it indicates the presence of blood. We put a small portion of the suspected gastric contents upon an object glass with a trace of common salt and a few drops of glacial acetic acid. If we now evaporate slowly to dryness there are formed small, brown, rhombic hemin

Fig. 71.—Hemin crystals.

crystals (see Fig. 71), which can be seen by the microscope. Of course, whenever we examine the contents of the stomach for blood, we should bear in mind that, if found, the blood may be due to the ingestion of underdone meat, preparations of hemoglobin, or similar substances.

To sum up briefly all that has been said, a careful examination of the stomach should proceed in the following manner: First, rinsing out of the fasting stomach early in the morning, to determine whether there may be stagnation or hypersecretion (marked HCl reaction). We should also notice whether there is any collection of mucus in the stomach when fasting. Secondly, we determine the position and size of the stomach by inflation. Thirdly, the stomach being empty, we administer a test breakfast; an hour later we examine the contents for hydrochloric or lactic acid. Fourthly, at noon we give a test meal, and seven hours later wash out the stomach, to determine its motor efficiency. If there is stagnation of the gastric contents, we test again for hydrochloric and lactic acids. Fifthly, microscopic examination of the gastric contents. Blood test. Finally, we scarcely need to repeat that in all important cases we ought not to be satisfied with a single examination of the stomach, but we should make certain of our results by repeated tests.

CHAPTER II

ACUTE GASTRIC CATARRH

(Acute Gastritis)

Ætiology and Pathological Anatomy.—The mucous membrane of the stomach not being open to direct examination, as is that of the mouth and throat, the existence of acute gastritis is mainly a matter of inference from our observations of other mucous membranes. When some harmful agency acts in a direct manner upon the mucous membrane of the stomach, we shall usually be right in supposing that there is a greater or less degree of genuine inflammation of the lining of the stomach, unless the organ in a simply functional way at once rids itself of the irritant—for example, by vomiting when the stomach has been "overloaded." If some noxious material acts long and continuously upon the mucous membrane, there will be abnormal changes not only in the blood vessels and the interstitial tissue, but also in the cellular elements of the mucous membrane and its glands. As yet, we know little about the minute degenerative changes in the specific secreting cells. The microscopic inflammatory lesions, which we may with great certainty assume to be present, are hyperemia and swelling of the mucous membrane, perhaps associated with an increase in the secretion of mucus, and with small hemorrhages here and there. If the irritant is insignificant the lesions will be mild and superficial (mild catarrhal gastritis). If the noxious agent is more powerful (for example, corrosive poison), it will give rise to a deeper parenchymatous inflammation, with peeling off of the mucous membrane and similar effects (severe toxic gastritis).

The mild catarrhal forms of gastritis are caused by simple chemical and mechanical influences, and perhaps by thermic irritation. They are most

often the result of errors in diet, such as the ingestion of too large an amount of food, or of food that is difficult to digest, unsuitable, highly spiced, or very acid. In the same class belong the acute indigestion following excess in alcohol, and the frequent derangement of the stomach from taking medicines; and also cases due to the accidental or willful ingestion of all sorts of injurious and poisonous substances. The severe cases of toxic gastritis are most often caused by the action of concentrated mineral acids and alkalies. In this class comes poisoning from sulphuric acid, nitric acid, hydrochloric acid, caustic potash, and caustic soda.

A special importance attaches to the ingestion of decaying substances. The incautious use of tainted meat or fish may be followed by relatively severe forms of acute gastric catarrh. The products of decomposition act as chemical irritants upon the mucous membrane; and the ferments and putrefactive agents likewise continue in activity after reaching the stomach, and thus contribute to produce the inflammation. The reason that this sort of gastric catarrh is not much more frequent than it is, is undoubtedly the presence of hydrochloric acid as a constituent of the gastric juice, because it has an antiseptic action.

It is universally assumed that a chill of the outer surface of the body may excite gastric catarrh, but there seems to be actual proof of this in but few cases. On the other hand, there can be no doubt that many cases of acute gastric catarrh, of apparently primary origin, are referable to infection. Infectious catarrh of the stomach may occur at times, particularly in summer, with especial frequency. As to the precise nature of the pathogenic germ, we do not as yet possess any certain knowledge.

The predisposition of certain individuals to gastric catarrh differs greatly. Some persons always have a "weak" stomach, and the disease is prone to attack feeble children, anæmic persons, fever patients, and convalescents from severe diseases, as well as chronic invalids who are ill-nourished. Enfeebled persons of this sort sometimes fall sick when vigorous and healthy individuals would entirely escape. In many of these cases of unusual predisposition to gastric disease, we may surmise that the secretion of hydrochloric acid is scanty, and that there may be a diminution in the motor power of the stomach. Each of these factors would, of course, render ingested irritants more harmful.

Symptoms.—The most constant subjective symptom is anorexia. In severe cases the very thought of food excites disgust. What the patient does eat tastes flat, and he is therefore very eager for piquant dishes, highly spiced or sour. Thirst is often present, and a feeling of dryness in the mouth.

The subjective gastric sensations are seldom those of marked pain; but sometimes there are attacks of pain which are in all probability due to cramp-like contractions of the muscular coat. The usual complaint is of constant pressure and fullness. Sometimes the patient is conscious of the peristaltic movements of the stomach. He has "rumbling" in the abdomen.

There is nausea, and often vomiting. In severe cases everything swallowed is at once rejected. The vomitus consists for the most part of undigested food (often ill-smelling), with which mucus, and sometimes bile, is mingled. Eructations of gas or liquid are frequent.

Physical external examination reveals little. The epigastrium may be

somewhat prominent as a whole, and may be tender on pressure. The tongue is almost always thickly coated and dry. The breath is usually disagreeable, and there is a persistent flat or bitter taste in the mouth. In ordinary cases of acute gastritis examination of the gastric contents is hardly necessary, but in the majority of the severe cases we find a marked diminution in, or entire absence of, the hydrochloric-acid reaction, and sometimes distinct delay in the motor activity of the stomach. Sometimes lactic acid and the fatty acids are present in large amounts.

In severe cases there is always considerable constitutional disturbance. The patient feels languid, and disinclined for any exertion; the pulse is moderately rapid, although it may exceptionally be slower than normal; the urine is usually somewhat concentrated, often it gives a marked indican reaction. There may be no fever, but not infrequently there is a moderate elevation of temperature with sensations of chilliness or heat. In rare cases we may observe an approach to the typhoid condition, with such nervous symptoms as headache, vertigo, and dullness. These cases were formerly termed "gastric fever," and they are probably most of them infectious, but yet the constitutional symptoms are probably only exceptionally referable to coincident constitutional infection. It is more probable that toxic influences are exerted by the abnormal products of the fermentation which takes place in the stomach. For example, Senator mentions sulphureted hydrogen as thus generated; and Litten has described several cases in which at first there were such dyspeptic symptoms as nausea, vomiting, flatulence, and a coated tongue, but which soon gave evidence, by restlessness, headache, great muscular weakness, and a gradual lapse into somnolence, of rather severe nervous disturbance in addition. The breath had a marked "acetone" odor; and, on adding chlorid of iron to the urine, a strong reddish color was developed (Gerhardt's [diacetic acid] reaction), so that it seems probable that an autointoxication had occurred somewhat resembling diabetic coma.

Chief among complications are the intestinal symptoms, which are frequently coincident with the gastric disorder. Constipation is the rule. There may be diarrhea. The gastric catarrh may by extension involve the duodenum and give rise to jaundice. Sometimes herpes appears upon the skin. This fact argues for the infectious nature of many cases of gastric catarrh.

It is evident from what has been said that acute gastric catarrh is by no means uniform in its ætiology, and the general course of the disease is also subject to great variations. Sometimes there are mild dyspeptic symptoms which vanish in the brief time of one or two days, while in other cases we have a rather severe disease associated with considerable constitutional disturbance, and lasting three to ten days. There is also great variation in the intensity of the various symptoms, particularly the vomiting. The progress of the case may be irregular and there may be relapses, but still the prognosis of primary acute gastritis is entirely favorable. The diagnosis is usually easy, although we should never omit to make an unprejudiced and careful examination into the general condition. If there is fever we should bear in mind the possibility of a mild typhoid (see Typhoid Fever).

Treatment.—If, at the beginning of the disease, there is reason to suppose that the stomach is loaded with undigested food, an emetic is indicated. If it is desired to avoid the irritative action of an emetic upon the gastric mucous

membrane, a subcutaneous injection of one sixth of a grain (gm. 0.01) of apomorphin may be given. Still more efficient, although disagreeable for the patient, is a washing out of the stomach by means of the stomach tube, particularly in severe cases of toxic or infectious origin; but of course, when the gastric walls may have been weakened by corrosive poisons, the stomach tube is contraindicated.

In most cases, however, emetics and lavage may be dispensed with. The treatment in such cases consists mainly in a strict regulation of the diet, allowing for a time nothing whatever, and then such food as thickened soups, iced milk, and toast. Of internal remedies, 10 to 15 drops of dilute hydrochloric acid in half a wineglass of water sometimes seems beneficial; while in other cases, in which there are sour eructations and vomiting, alkalies are to be given. We may order as much bicarbonate of soda or as much Carlsbad salts as will rest on the point of a penknife, or Ems water. The so-called stomachics and bitters are also often prescribed, for example, compound tincture of gentian or tincture of rhubarb. Another favorite remedy is resorcin, of which 4 gr. dissolved in some aromatic water may be given every two hours. If the vomiting is obstinate, relief may be got from bits of ice or sips of cold Seltzer water. In severer cases we must resort to narcotics, such as opium, cocain, and chloroform, administered internally.

If there is decided constipation, an enema must be employed, or such laxatives as calomel, Epsom salts, or rhubarb.

CHAPTER III

CHRONIC GASTRITIS—CHRONIC CATARRH OF THE STOMACH

Ætiology.—The same causes which excite acute gastric catarrh, if often repeated, lead at last to chronic catarrh of the stomach; but, at least in adults, infectious influences are seldom prominent in the causation of the disease. In many cases it is due chiefly to the chemical and mechanical irritation of an unsuitable diet long continued. By far the most frequent—indeed, to speak more correctly, the only frequent—form of chronic gastric catarrh in adults is due to hard drinking. The habitual use of distilled liquors is more influential in this regard than excess in wine or beer. The harm done by alcohol may be reënforced by other dietetic causes. We have often met with genuine chronic gastritis in extremely destitute persons, who for a long time had to eat food that was insufficient, bad, or even decayed. It should also be pointed out that abnormal conditions of the mouth, and, particularly, dirty or carious teeth, may occasion dyspeptic and catarrhal conditions. Habitual excess in smoking is also said sometimes to occasion chronic gastritis; and finally, it is probable that the bad habit of rapid eating with imperfect mastication leads at last to gastric disturbance. As is the case with all external irritants of this sort, the results are influenced by individual differences in the resisting powers of the affected organ, and personal predisposition to disease plays a certain part in the development of chronic gastric catarrh. Often this predisposition seems, to a certain extent, a matter of family and heredity.

Chronic gastric catarrh is not always a primary affection. It may be secondary to some other disease. In particular, all diseases associated with portal congestion are apt to lead to secondary catarrh of the stomach. This includes hepatic cirrhosis and hepatic syphilis. Again, the gastric catarrh associated with chronic cardiac, pulmonary, or renal disease is in part to be regarded as due to congestion. On the other hand, however, we should always consider how manifold are the other conditions, associated with all sorts of chronic diseases, which may lead to chronic gastric disturbance. We may mention, for instance, anæmia, muscular insufficiency, and autointoxication.

Pathology.—The macroscopic changes in the gastric mucous membrane are in most cases very moderate. Usually it is thickened and coated with a layer of tough, grayish-white mucus, in which is suspended a greater or less amount of detached epithelium. The membrane is brownish red, unless rendered gray by excessive pigmentation. Under the microscope we find a well-marked infiltration of the interstitial tissue with round cells, and well-marked parenchymatous changes, particularly extensive degeneration of the glandular cells. All these changes are especially marked in the pyloric portion of the stomach. If the catarrh has lasted a long time there may be still further changes in the mucous membrane. In many cases it appears smooth and atrophic. The glandular layer of the stomach may at last be almost destroyed, and the muscular and submucous coats share in the atrophy, while the organ as a whole is usually dilated. In other less frequent cases there is a marked growth of interstitial connective tissue, with resultant contraction. In this variety, also, the glandular layer atrophies, the stomach walls grow firmer, and the whole organ shrivels (cirrhosis of the stomach). Contrasting with these forms which lead to atrophy or sclerosis, there are others which occasion a hyperplasia of the mucous membrane. Its inner layer is thickened and mammillated (*état mamelonné*), and there may be actual polypi formed. The hyperplasia in these cases affects mainly the gland tubes of the mucous membrane, but there is also a considerable thickening of the submucous coat.

Symptoms on the Part of the Stomach.—The symptoms of chronic gastric catarrh are the same dyspeptic symptoms which we meet in various combination and degree in all diseases of the stomach. They include disturbance of the appetite; pressure or pain in the region of the stomach; a bad taste in the mouth; abnormal sensations in the throat; and finally, motor phenomena, including rumbling, eructations, and vomiting.

The appetite is usually impaired in chronic gastric catarrh. There is sometimes a moderate appetite, but it is soon changed to a feeling of repletion upon the ingestion of even a slight amount of food. In other cases there is actual dislike of any form of nourishment; the patient eats little, and prefers highly spiced, piquant dishes. There is often a persistent bitter, flat, disagreeable, or otherwise abnormal taste in the mouth.

Subjective sensations in the region of the stomach are rarely entirely absent. As a rule, there is a feeling of fullness or pressure and of dull pain. These troubles may either be constant or occur after meals. Sometimes, especially after errors in diet, there may be actual pain. This is termed cardialgia or gastralgia, and is probably due in most cases to cramp-like contrac-

tions of the muscles; but in general, habitual and severe pain does not belong to the clinical picture of simple catarrh.

A very frequent and annoying symptom, which is particularly apt to come on after eating, is the eructation of gas, usually air, but sometimes gases which are formed in the stomach by the processes of decomposition. Such gases have a disagreeable odor. The eructations may bring up, also, some of the fluid contents of the stomach. If there is a bitter taste associated with it this is usually occasioned by peptones, but sometimes perhaps by bile (*vide infra*). Acid eructations indicate excessive acidity of the gastric contents, ordinarily due to hydrochloric acid, but exceptionally to lactic acid (*vide infra*), or to the fatty acids, which are recognizable by their odor. The burning feeling in the pharynx and lower down, which is occasioned by acid eructations and which is usually termed heartburn, is almost always caused by an excess of hydrochloric acid in the gastric contents, and consequently is not a frequent symptom of genuine chronic gastric catarrh (*vide infra*).

In many cases the feeling of nausea increases, especially after eating, to actual vomiting, but in chronic gastric catarrh vomiting is not very frequent, and if it occurs it is the result of some special cause, such as errors in diet. There is this exception, that in the chronic gastric catarrh of drunkards a peculiar form of vomiting is very common. This is called morning vomiting. By this is understood a fairly regular occurrence of vomiting in the morning before breakfast, with the ejection of considerable amounts of watery mucus, usually alkaline. This vomiting is due not only to the chronic catarrh of the stomach, but also to the chronic catarrh of the pharynx, which latter occasions a great tendency to gagging—e. g., when rinsing out the mouth. The vomitus consists not only of mucus from the stomach, but in large part, also, of a mixture of saliva and mucus from the mouth and throat. In the other forms of chronic gastric catarrh the vomitus consists often of ill-digested food and more or less mucus. If there are vigorous efforts at vomiting and retching, the vomitus may contain also some bile and small amounts of blood, which have no serious significance.

The external examination of a patient with chronic gastritis shows little unusual. The general nutrition, of course, is often impaired, although in drunkards and gourmands an abundant fat layer persists for a long time. The tongue is usually coated, especially in its center, while its sides and tip are red. A thickly coated tongue and salivation are both of them due rather to accompanying stomatitis and pharyngitis—complications which are present in the case of most drunkards, and sometimes also in excessive smokers. Examination of the epigastrium shows often a considerable prominence of the stomach and tenderness on pressure, but the only means of obtaining an actual insight into the abnormal process is a careful investigation by means of the stomach tube. This should not be omitted in any case of chronic gastritis of any severity, especially as the results of it furnish the only true guide for a rational treatment of the disease. If we examine the gastric juice after the manner already described, we shall find that free hydrochloric acid in chronic gastritis is scanty (below 0.1 per cent), or even absent. Its complete absence is particularly common in chronic alcoholic catarrh, and also in cases of chronic gastritis in which there is atrophy of the mucous membrane. In the latter case pepsin, also, is often absent. With regard to

abnormal acids, we may find lactic acid, but seldom in large amounts, because in simple chronic catarrh there is scarcely ever much stagnation of the gastric contents. If there are abnormal processes of decomposition, we find acetic acid, butyric acid, and volatile fatty acids, but these are very rarely present.

The demonstration of an excessive production of mucus in the stomach is of great diagnostic importance. We have mentioned that sometimes the vomitus contains a distinct admixture of mucus, but the only way of forming an accurate opinion with regard to the amount of mucus in catarrh of the stomach is by siphonage, especially in cases in which vomiting is absent or infrequent. If we examine the stomach when fasting we shall, in many cases, find at every trial an abundance of mucous fluid with scarcely any other ingredients. In other cases there is more apt to be an abundance of mucus after a meal—either in a viscid, ropy fluid, or in separate transparent masses of varying size. Excess of mucus in the stomach is usually associated with anacidity, but there are a few exceptional cases in which it is associated with increased secretion of hydrochloric acid (gastritis acida; acid catarrh of the stomach. Boas, Riegel, and others). We must avoid confounding secretion from the mouth and pharynx that has been swallowed with mucus produced in the stomach. The latter is intimately mixed with the food, if there be any.

The motor function of the stomach is seldom much disturbed in chronic gastritis. Often it is surprising how rapidly the stomach empties itself; but there may be delay, partly because digestion is impaired and partly because there is a gradual development of weakness and atrophy of the muscular coat of the stomach, analogous to the weakness of the muscles of the vocal cords in chronic laryngitis. If food is long retained in the stomach, there is probably some other disease than simple catarrh. The size of the stomach in chronic catarrh may be somewhat increased, but this has no special importance.

Symptoms on the Part of Other Organs. Course of the Disease.—Of the other organs, the intestine most frequently suffers in chronic gastric catarrh. In almost all cases of chronic gastric catarrh the bowels are irregular. Habitual constipation is the rule, but sometimes there is diarrhea. If much gas is generated in the stomach, the intestinal canal often becomes implicated, and tympanites and flatulence develop.

There has been great stress laid, particularly in recent times, upon the frequent occurrence of nervous disturbance in association with chronic catarrh of the stomach. This takes the form of hypochondriasis and depression of spirits in association with all sorts of other nervous symptoms, including pressure in the head, headache, dullness of mind, and dizziness (*vertigo e stomacho laeso*); but, while we may admit that the disease of an organ which, for many human beings, is the main source of happiness, may well put the patient out of sorts, yet there is no doubt that many earlier reports about nervous disturbances were due to a confusion of chronic gastric catarrh with nervous dyspepsia (*vide infra*). Genuine chronic catarrh of the stomach, as such, has no peculiar relation to nervous disease, except in the rare cases in which certain unusual symptoms develop, not improbably from the absorption of toxins.

When the catarrh is of any duration and severity, the general nutrition is apt to be seriously impaired. The diminished appetite and the imperfect digestion and absorption of what is eaten contribute to produce a gradual

and considerable loss of weight. The fatty and muscular tissues atrophy. The skin grows dry and harsh, and usually has a dirty pale color. In rare cases of extreme atrophy of the mucous membrane (probably almost always combined with atrophy of the intestinal mucous membrane) there are developed symptoms like those of progressive pernicious anæmia (*q. v.*).

Individual cases differ greatly in the combination of symptoms they present and in their course. The anorexia, gastric oppression, eructations, vomiting, and other important disturbances already mentioned, exhibit the greatest diversity in their intensity and their grouping. In the milder cases, loss of appetite and moderate local uneasiness may be the only symptoms. Frequent vomiting is confined to the severer cases. The disease often lasts for years, especially if the patient neglects himself. In most cases there are frequent remissions and exacerbations, usually dependent upon external causes.

The disease is not intrinsically fatal except in the above-mentioned rare cases of complete atrophy of the mucous membrane, but the general debility consequent upon it may indirectly shorten life.

Diagnosis.—If we except the self-evident cases of chronic alcoholic catarrh, the only proper way of making a diagnosis of chronic gastritis is by means of a careful examination of the gastric contents, along with a consideration of the associated conditions. If a patient has for some time been suffering from the ordinary gastric symptoms of anorexia, gastric oppression, eructations, and vomiting, we must first determine whether we have to deal with a primary disease of the stomach or with a secondary disturbance referable to such conditions as cardiac disease, renal disease, or pulmonary tuberculosis. If the indigestion is not symptomatic we must then consider in order each of the primary diseases of the stomach. If there is no indication of ulcer or carcinoma, we have remaining actual gastritis, nervous dyspepsia (*q. v.*), and a dislocation of the organ (gastroptosis). One important factor is the general condition of the patient—for instance, if there is neurasthenia or the like. No actual conclusion can be reached, however, except by examining the contents of the stomach. We cannot assume that there is a chronic gastritis unless there are direct objective changes to be demonstrated, chief among which is an abnormal production of mucus, associated with a change in the amount of hydrochloric acid in the gastric juice. This change is in most cases a diminution, but exceptionally an increase of the amount secreted. We wish, in this connection, to emphasize once more the statement that genuine chronic gastritis, except in drunkards, is by no means a common disease and is much less frequent than nervous dyspepsia.

Treatment.—If the disease seems to be merely symptomatic—the result, for instance, of venous stasis due to chronic cardiac, pulmonary, or hepatic disease—our efforts must, of course, be directed chiefly to the relief of the original trouble. We must also take into consideration all other causative influences, such as neglected or decayed teeth, improper mode of life, and unhealthy occupation.

The direct treatment of chronic gastric catarrh must always begin with a regulation of the diet. Such vague injunctions as "to be cautious" or "to avoid indigestible articles of food" are useless. The patient must have a perfectly definite bill of fare prescribed for him; nor can any universal diet, suitable for all cases, be drawn up. In each individual instance the individual

circumstances must be considered. The personal experiences of the patient himself are by no means to be disregarded. One man may be quite unable to digest what is well borne by others, and *vice versa*.

In the first place, certain foods must be utterly forbidden to such patients as do not themselves avoid whatever disagrees with them. All articles must be prohibited which may irritate the mucous membrane, either mechanically or chemically. This includes all the coarser sorts of vegetables or fruits containing a large proportion of indigestible cellulose, and all dishes that are very sour, strongly salted, or highly spiced. Potatoes, farinaceous food, and all substances composed mainly of hydrocarbons must also be interdicted, because almost all the abnormal fermentative processes, the evil consequences of which have already been considered, are promoted by the hydrocarbons. Fat is also harmful. It impedes digestion by protecting the contents of the stomach from the action of the gastric juice in a purely mechanical way, and then, being changed into the fat acids, it causes sour eructations and pyrosis. Yet we must not go too far in forbidding starches and fats. Particularly if the patient is emaciated it is advisable to give such fatty substances as good butter and cream in cautious amounts, and often the result is satisfactory. In general, we should not forget that with regard to diet the skill of the physician often lies more in what he permits than in what he forbids. The limitation or withholding of alcoholic beverages is an important point. As we have already mentioned, the great majority of cases of chronic catarrh are the direct result of excess in alcohol—the most important factors being chemical irritation and mechanical distention of the mucous membrane. Moreover, the experiments of Fleischer and others have shown that alcohol prolongs and hinders the process of digestion. In every severe case it is better to forbid alcoholic beverages entirely, and ordinarily such an injunction is much better obeyed than advice to use them sparingly. In mild cases we admit that small portions of beer and wine may be permitted unhesitatingly, especially if the patient himself experiences no feeling of discomfort from them, but rather an improvement in his appetite.

In determining what the patient may be allowed to eat, we are to consider, as already mentioned, his own personal experience as well as our more general knowledge. An intelligent patient will often be himself the best judge of what agrees or disagrees with him. The following foods are very easy to digest: milk, soft-boiled or raw eggs, broths (especially veal and chicken broths), and certain artificial preparations, chief among which stand meat juices, peptones, sanatogen, somatose, and many others. It must be confessed that patients soon tire of these articles. The brain and sweetbread of calves are easily digestible; also birds, such as pigeons, chickens, and partridges, thin shavings of raw beef or raw ham, rice, potato *purée*, etc. Gradually we may proceed to somewhat heartier food—veal, game, roast beef, trout, and light farinaceous dishes. The worse the symptoms are in any case, the more strict must we be in regard to diet. For drink, besides water or Seltzer water, very weak tea, cocoa, chocolate, and water reddened with claret are allowable.—Shall we permit coffee? This is a question which must be answered according to the individual experience of the patient. Coarse bread is to be forbidden. Ordinary white bread, toasted, if it seems desirable, may be allowed in moderate amount, also rusks.

Solid articles of diet must be finely cut up and well chewed before being swallowed. The food must not be very hot or very cold. It is sometimes advantageous to take more than three meals a day, each one being proportionally smaller. Other patients relish their food better if the intervals between eating are prolonged. It should be added that excessive smoking is harmful in chronic dyspepsia.

There are other special indications to be met. In all severe cases by far the most efficient mode of treatment consists in methodical washing out of the stomach. This is especially true when there is excessive production of mucus, or a tendency to decomposition of food in the stomach. In this way we prevent the accumulation of any great amount of undigested food. We remove the excreted mucus and the products of abnormal fermentation and decomposition, and, moreover, we may perhaps exert a direct and favorable influence upon the mucous membrane. In such cases we are accustomed to rinse out the stomach daily, and the best time for this is in the morning before the patient has had breakfast. Fresh fluid must be poured in and emptied out rapidly, until the stomach is freed from all foreign material, especially mucus. We use for the rinsing fluid a weak solution of hydrochloric acid (one to two per mille), or a one-per-cent solution of either common salt or bicarbonate of soda; and, if there is much mucus, a favorite remedy is very dilute limewater, two to four tablespoonfuls of liquor calcis to a liter (quart) of water. If fermentation is going on, we employ weak solutions of hydrochloric, boric, or salicylic acids, or resorcin. It has also been recommended to spray or douche the mucous membrane of the stomach, for which purpose stomach tubes are employed with numerous small openings, and such fluids are chosen as weak solutions of alum, tannin, and condurango.

If we now turn to a consideration of the internal remedies used in chronic gastric catarrh, it is possible to decide with regard to their employment in a much more intelligent fashion than heretofore, because in all severer cases we make a more accurate investigation of the digestive process. If the examination of the gastric juice shows a diminution of hydrochloric acid, this may be artificially supplied. We prescribe thirty to sixty minutes after each meal, 10 to 15 drops of dilute hydrochloric acid in half a glass of water. The benefit to be thus derived must not be overestimated. In appropriate cases, however, its influence is favorable. If there is no pepsin in the gastric juice we may prescribe pepsin as well as hydrochloric acid. Pancreatin and papain are also recommended, but their usefulness is very doubtful. Pankreon seems to me more active. The popular wines of pepsin are not to be recommended, because of the alcohol in them.

Alkalies have long been employed as well as hydrochloric acid, and often with good results, although the most common form of chronic gastric catarrh has a less than normal secretion of hydrochloric acid, and consequently the use of alkalies would seem to be contraindicated; yet experience shows them to be useful. Probably their beneficial effect is that they neutralize the abnormal acids, promote the emptying of the stomach into the duodenum, dissolve the mucus, and finally stimulate the secretion of the gastric juice. This has been experimentally demonstrated with regard to carbonate of soda, common salt, and carbonic acid. From what has been said it is evident that in chronic gastric catarrh alkalies are not usually given during digestion, but when the

stomach is empty and before meals. As is well known, the alkalies are prescribed more especially in the form of alkaline mineral waters, which the patient drinks at home, or at the various health resorts. The springs of Carlsbad possess the greatest reputation. Those at Ems, Kissingen, Tarasp, and Vichy also deserve to be mentioned as health resorts for patients with gastric trouble. A good portion of the benefit at these places is, to be sure, dependent upon the fact that many patients are much more apt to follow a strict regimen when they make use of a particular "cure" than when they remain at home. It is self-evident that large doses of bicarbonate of soda are indicated in that form of chronic gastritis which is associated with hypersecretion.

If the character of the vomitus, or gastric contents obtained by rinsing, indicates abnormal fermentation, we may not only wash out the stomach (*vide supra*), but also try certain antifermentative remedies. First among these is hydrochloric acid in rather large doses, and we also give salicylic acid (in powders, containing 8 gr. [gm. 0.5]); creosote (two or three 0.5-gr. pills daily [gm. 0.03]); benzine (20 drops in capsules or in milk), and similar drugs. As a rule, however, the last-named remedies are seldom employed.

The best drugs to stimulate the secretion of gastric juice are the bitters. It is this property which has earned them the name of stomachic tonics. Compound tincture of gentian, tincture of nux vomica, tinctura amara, and tinctura calami, P. G., quassia and columbo—these are much employed. In general, however, they are not very efficient. An excellent tonic in many of these cases is condurango bark. A decoction may be made (15 parts to 200 of water); or we may employ the fluid extract, giving a teaspoonful in water two or three times a day.

A few remedies remain to be mentioned, which are said to exert a direct beneficial influence upon the catarrh, and which many physicians extol highly. Their efficacy, however, is somewhat problematical. We refer chiefly to subnitrate of bismuth, sulphate of zinc, and nitrate of silver. (See prescriptions in the Appendix.)

Certain symptoms may demand especial treatment, such as vomiting. It will usually yield to regular and persistent washing out of the stomach. Other remedies are small bits of ice and minute doses of opium or chloral. Potassium bromid and chloroform, internally administered, may also be tried.

Violent gastralgia requires narcotics, such as morphin and opium. The best external applications are poultices or wet compresses. "Sour stomach" may be relieved by a pinch of bicarbonate of soda, or of calcined magnesia. Persistent anorexia may yield to the bitters mentioned above, or to small doses of quinin or compound tincture of cinchona. If the bad taste in the mouth is annoying, the mouth should be frequently rinsed out with Seltzer water, a one-per-cent solution of carbolic acid, or five drops of tincture of myrrh to a glass of water. For habitual constipation, enemata, or the various mineral waters, are good; also Carlsbad salts. In obstinate cases pills of rhubarb or aloes may be employed. Still, we ought never to forget that the infrequency of the stools is often merely a natural consequence of the scanty diet, and that it is therefore possible to do harm with our purgatives. Iron is often prescribed for the concomitant anæmia; but it should be employed cautiously, for it is often ill borne in gastric disease.

CHAPTER IV

PHLEGMONOUS GASTRITIS

(Purulent Inflammation of the Stomach)

Purulent inflammation of the stomach is very rare, and little is yet known about it. In most cases no special causes for it have been ascertainable. It is occasionally one of the symptoms of grave pyæmic or puerperal inflammation. We have seen one case in which suppuration of the pharynx led to an acute phlegmonous inflammation of the submucous coat of the œsophagus, also involving a large portion of the stomach, with a fatal termination.

Two forms are distinguished—a diffuse and a limited variety. The latter is equivalent to gastric abscess. The submucous layer is almost invariably the chief seat of suppuration. From this starting point the process invades the muscular and serous coats on the one hand, and the mucous membrane itself on the other—the usual result being multiple sieve-like perforations inward or outward.

The usual symptoms are violent gastric derangement, with pain and vomiting, high fever, and the indications of constitutional infection, namely, headache, delirium, and general prostration. Sometimes the disease is quickly fatal, sometimes it runs a more chronic course. The few cases in which recovery has been reported are somewhat obscure.

The disease can never be diagnosticated with absolute certainty. Treatment must be purely symptomatic. Ice, both internally and externally, and the narcotics, are chiefly employed.

CHAPTER V

GASTRIC ULCER

(Simple or Round Ulcer of the Stomach)

Ætiology.—Since Cruveilhier gave the first accurate description of gastric ulcer, numerous explanations of its occurrence have been propounded. Even yet there is no universally accepted view. We may, in general, hold to the assertion that the development of an ulcer is preceded by a local disturbance of nutrition, or necrosis of the gastric mucous membrane, and that then the enfeebled or necrotic tissue is dissolved by the gastric juice—i. e., digested ("peptic ulcer"). But it is not known what are the special causes which occasion the primary damage to the tissue, nor why the loss of substance when it occurs is not at once healed, but extends in width and depth. There is, indeed, no lack of theories to solve these questions, and pathologists have made many experiments with regard to the pathogenesis of gastric ulcer.

The explanation of the fact that the normal gastric mucous membrane is not attacked by gastric juice lies probably not so much in the presence of the alkaline blood flowing through the membrane as in the intrinsic vital

resisting powers of the normal living cells. If the cells are in any way damaged there occurs at once an autodigestion at that spot. Damage of this sort may be produced experimentally in different ways; for example, by artificially produced embolism of the smaller arteries of the stomach, and by wounds, including bruising, burning, and cauterization of limited portions of the mucous membrane. In all such cases a peptic ulcer is formed, but almost invariably this promptly heals, or, at any rate, it shows no tendency at all to extension. If, as a symptom of severe gastritis or portal congestion, there are small hemorrhages into the mucous membrane, they result in so-called hemorrhagic erosions; but these again are limited to the small spot destroyed by the hemorrhage.

Virchow assumed that ordinary round ulcer of the stomach in man is, in most cases, the result of the plugging of the small vessels with a thrombus or embolus, because of various diseased conditions of their walls, but this supposition remains entirely without proof. One fact that tends to contradict this view is the appearance of ulcer in young individuals without any signs of cardiac or vascular disease. Only in exceptional cases does a gastric ulcer develop in older persons apparently on an arteriosclerotic basis. Other investigators thought of the possibility of accidental injuries, and, perhaps in some few cases, even, of external trauma (burns, etc.). Such suppositions may be correct, although they, again, in most cases are incapable of demonstration. Then we have still to meet the second above-mentioned question: Why, in all such cases, the little erosions, if they are formed, do not immediately heal, as the experimentally produced ulcers almost invariably do. Lately the attempt has been made to settle this difficulty by pointing out that in ulcer of the stomach the acidity of the gastric juice is shown to be abnormally great. This hyperacidity is assumed to exist even before the development of the ulcer, and in a certain degree to cause a predisposition for its development, as well as to explain the unwillingness of the ulcer to heal and its proneness in many cases to extend farther, to spread. If a gastric ulcer is artificially produced, the process of healing may be considerably delayed by introducing solutions of hydrochloric acid into the stomach. It is very possible that hyperacidity of the gastric juice is a factor in the ætiology of ulcer, but the whole matter is by no means easily intelligible. We remain uncertain as to what is the primary cause of the ulcer, and it has not yet been proved that hypersecretion and hyperacidity exist before the formation of the ulcer. We might also believe that the excessive acidity is secondary to the sensory irritation occasioned by the raw spot. In a word, our present knowledge of the development and extension of gastric ulcer is vague.

Gastric ulcer occurs but seldom in childhood. It is most often seen between the ages of eighteen and thirty. The cicatricial stenosis of the pylorus and other sequelæ are often seen in still older individuals, between thirty and forty-five years; but in most of these cases the disease probably originated much earlier. In still later life gastric ulcer is rare. In general, the disease seems to be more frequent in the female sex than in the male; but if we count only the absolutely demonstrated cases, the difference is not a very great one. The view that ulcer of the stomach attacks by preference anæmic and chlorotic girls is very general, but to the author seems much exaggerated. The probable explanation is that gastric disturbances in chlorotic persons are often

ascribed to gastric ulcer without sufficient reason. Taking into consideration only the absolutely certain cases, the author's experience does not show any especially close connection between chlorosis and gastric ulcer. Yet it must, of course, be confessed that indubitable cases of gastric ulcer occur in chlorotic girls.

Pathological Anatomy.—The ulcer is usually approximately round or oval. Its borders are sharp; the walls often slope inward, giving the ulcer the form of a shallow funnel. The base of the ulcer is almost always perfectly clean, so that in microscopic sections we see the ends of the gland tubes remaining unchanged, and reaching out into the surface of the ulcer. After an ulcer has lasted some time, a reactive inflammation develops around the necrotic area, leading to the formation of connective tissue and cicatrization. If superficial, the ulcer does not extend farther than to the muscular coat, but it may be deep enough to expose the serous membrane, or even to perforate it (*vide infra*). The size varies greatly. Some are hardly as large as a pea; others may measure 10 to 15 cm. in their greatest diameter. As to position, most of them are found near the pylorus. They attack the posterior wall of the stomach, particularly the neighborhood of the lesser curvature, far more frequently than the anterior wall, but the greater curvature is not infrequently the seat of an ulcer. As a rule, we find but a single ulcer, although exceptions to this statement are not very rare.

If an ulcer of any size heals, a scar is formed, with radiating lines and often of considerable size; smaller superficial ulcers heal with a smooth scar. Cicatricial contraction may alter the shape of the stomach considerably. If a deep constriction is formed around the middle of the stomach, we have what is called the hour-glass shape. Scars of pyloric ulcers are of the greatest clinical importance, because they lead to cicatricial stricture of the pylorus, with resultant dilatation of the stomach.

If the gastric ulcer extends to the serous membrane, the final result may be perforation into the abdominal cavity and general peritonitis, unless the stomach previously becomes attached at the point threatened to some neighboring organ, because of adhesive inflammation. The ulcers being usually on the posterior wall of the stomach, it is oftenest the pancreas to which the stomach becomes adherent. In other instances it is the liver, transverse colon, diaphragm, or spleen. If adhesions are formed before the perforation of a gastric ulcer, there may be circumscribed peritoneal abscesses. These are situated with comparative frequency between the upper surface of the liver and the diaphragm, or between the stomach and the diaphragm (subphrenic abscess). Again, after such adhesions there may be perforation into the pleural cavity, the transverse colon, the pericardium, or the lungs.

The ulcer may cause erosion of a blood vessel, and thus give rise to one of the most important symptoms of the disease, namely, gastric hemorrhage.

Clinical History.—There may be absolutely no symptoms. It is not a rare thing to find at autopsies a still active ulcer of the stomach, or the cicatrix left by one, in subjects who never had during life any gastric disturbances whatever. Nor is it very exceptional for a person suddenly to exhibit grave symptoms, such as gastric hemorrhage, or peritonitis due to perforation, when there has been no reason previously to apprehend the existence of an ulcer.

In other instances the ulcer does, indeed, give rise to symptoms, but they

are not sufficiently characteristic to point to the correct diagnosis. Usually the gastric symptoms are long continued, but comparatively slight. They consist, for instance, of a sense of oppression or slight pain in the epigastrium, eructations, and occasional vomiting. In these cases, also, grave symptoms consequent upon the ulceration may suddenly arise.

In a third class of cases there are symptoms which are, to a certain extent, characteristic, and lead with more or less definiteness to the true diagnosis. These "symptoms of ulcer" are chiefly a peculiar epigastric pain, which is usually intermittent, and vomiting, or, what is yet more distinctive, the vomiting of blood, or hematemesis; also the signs of an increased secretion of hydrochloric acid in the gastric juice (hyperchlorhydria and hypersecretion). These symptoms and their diagnostic value we must now consider in detail.

Pain in the stomach is one of the most frequent symptoms of round ulcer. Its forms are very diverse. Often the patient complains only of a diffuse, painful sensation of pressure referred to the entire region of the stomach. This may be uninterrupted, or it may occur only after meals, or after excessive exertion, or as the result of some other special cause. This sort of pain is the least diagnostic of any, inasmuch as exactly similar sensations may be caused by other chronic disorders of the stomach. More characteristic of ulcer is a decided cardialgia, or, more correctly, gastralgia—that is, a very violent pain, coming on at intervals like neuralgia. It is described as "cutting," "tearing," "boring," and the like. These attacks of pain occur at various times, but oftenest after eating, and particularly after the ingestion of a large amount of food, or of food of a rather coarse sort. Often they occur with tolerable regularity a definite time after eating, say half an hour or an hour. This is partly to be explained by the beginning of the expulsion of food through the pylorus, and partly by the acme of the secretion of acid being due at this time. The pain is felt chiefly in the epigastrium, but not infrequently it extends toward the umbilicus, backward toward the vertebræ, into the thorax, or even into the upper extremities. In many instances a marked sensation of thoracic oppression accompanies it. A change of position may sometimes affect the severity of the pain. It is sometimes observed that the patient, when lying upon his right side, feels violent pain, which is at once relieved by changing to the left side, probably because the ulcer is located near the pylorus. An attack of cardialgia may last for a few minutes or for several hours. A third variety of pain may be observed in gastric ulcer. The suffering may be localized in a very limited area. Such pain is thought to be due to irritation of the floor of the ulcer by food, or to its edges being pulled upon during the movements of the organ. It generally comes on after eating, and ceases if the stomach is perfectly quiet. In position this pain is generally epigastric, but sometimes it is umbilical, or even, now and then, more toward the back. In many cases of gastric ulcer there is also tenderness on pressure in a quite sharply defined area and at one particular spot. Most authors regard the accurately localized pain as the most nearly pathognomonic; but it must be said that it is decidedly the least frequently exhibited of any. Transitional forms and combinations of the various kinds of pain are often observed. Two things may be said to be characteristic in all the sorts of pain connected with ulcer of the stomach: the localization of the pain each time in the same spot, and the relation of the pain to the ingestion of food. Com-

plete bodily rest is beneficial; external pressure upon the epigastrium usually increases the pain.

Vomiting is a frequent symptom in gastric ulcer, although it may exceptionally be absent or nearly so. Ordinarily, vomiting occurs, like the cardialgic attacks, after eating, and particularly after partaking of indigestible dishes. The vomitus in such cases consists mainly of partially digested food. It usually has a strongly acid reaction. If there is hypersecretion there may sometimes occur vomiting of a strongly acid fluid, not containing much that has been swallowed. If the ulcer has led to stenosis of the pylorus, the way in which the vomiting occurs is very characteristic. We shall describe this form of vomiting later, in detail. Vomiting is of the greatest diagnostic importance when blood appears. This may be intermixed in greater or less amount with the other contents of the stomach, or it may be vomited as pure blood (hematemesis). A gastric hemorrhage demonstrated by the vomiting of blood is the most valuable diagnostic sign of gastric ulcer. If there is no hemorrhage at all, the recognition of gastric ulcer is almost always rather difficult and uncertain.

Hematemesis is frequently the symptom which first leads the patient to apply to a physician. Up to this time he may have felt perfectly well, or, although there may have been some gastric derangement, he has not thought anything of it. The patient suddenly becomes faint, perhaps while he is pursuing his regular occupation, or it may be at night. He feels dizzy, and everything looks black. Then he has nausea, and finally is obliged to vomit. The vomitus is either pure blood, or a mixture of blood and food. It is partly coagulated, and often has a rather dark or blackish color, like tar. This change in color, as well as the coagulation, is due to the action of the gastric juice. The hemoglobin is transformed by the action of the hydrochloric acid into hematin. If the blood, before it is vomited, has remained for some time in the stomach, the vomitus has the appearance of "coffee grounds," and no longer contains unchanged red blood corpuscles (for the chemical demonstration of blood, *vide supra,* page 517). The amount varies greatly in different cases: there may be a quart or more. Sometimes there is a single hemorrhage, but not infrequently blood is repeatedly vomited either at short intervals or on successive days. Part of the blood escapes through the pylorus, so that, after a profuse gastric hemorrhage, blood is sure to be found in the stools. In them it is black and tarry. Exceptionally it happens that all the blood, beyond what is absorbed from the intestinal canal, passes off *per anum,* so that none whatever is vomited. In such cases it is often a difficult matter to locate the hemorrhage. If there is a sudden faintness with pallor, and without vomiting, but followed by black fecal discharges, we should always think of the possibility of a gastric hemorrhage. Sometimes the diagnosis of a gastric ulcer becomes probable if small quantities of blood can be demonstrated chemically in the stools, when the patient has been on a diet free from hemoglobin (so-called occult gastric hemorrhage).

The consequences of gastric hemorrhage depend, of course, chiefly on the amount of blood lost. Sometimes, although fortunately rarely, a large blood vessel is eroded and the patient dies. This event may be sudden, or it may occur more gradually under the influence of repeated hemorrhages and after a few days, during which all the symptoms of acute anæmia are exhibited. On

the other hand, the loss of blood may be so insignificant as to produce no especial symptoms. In most instances life is not actually threatened, but yet the signs and results of a more or less marked general anæmia are clearly visible.

In such cases the patient feels extremely exhausted, and at once takes to his bed. He has also all the subjective symptoms of cerebral anæmia. There are vertigo, tinnitus aurium, specks before the eyes, frequent gaping, and sometimes headache. To assume an erect posture aggravates the disturbance. There is usually excessive thirst. Now and then a temporary amaurosis has followed an excessive hemorrhage.

Objectively, we notice at once the excessive pallor of the skin, particularly of the face. The lips and conjunctivæ are also blanched. The pulse is rapid, and often ill sustained. For some days there may be anæmic murmurs over the heart, and there is a distinct sound to be heard in the femoral arteries. A moderate rise of temperature is very common, probably due to the absorption of the blood decomposing in the intestines. This is known as anæmic fever. The urine is pale, and usually rather abundant. Its specific gravity is not infrequently relatively high, namely, 1.015 to 1.020. All these symptoms are directly referable to the loss of blood, and they will be discussed with greater detail in the section on anæmia.

If the hemorrhage is not repeated, the patient gradually regains his strength. To be sure, the pallor usually persists for a long time, but the disagreeable symptoms gradually abate. When gastric discomfort has existed previously to the hemorrhage, it often disappears entirely after it—a circumstance which is probably due in part to the excessive caution of the patient thereafter. At the end of a few weeks the patient often feels perfectly well again: and, indeed, recovery is not infrequently complete and permanent. In other cases, however, the symptoms of ulcer return, sooner or later.

The third group of symptoms in ulcer of the stomach is furnished by an examination of the gastric contents with the aid of the stomach tube. These are of extreme importance. Earlier, when the soft tube now universally employed was not in use, we feared to introduce a tube into the stomach in case of ulcer, and even now we should not do it, in case of hematemesis, if it can be avoided, until some weeks have elapsed; but with this exception abundant experience has shown that we do not need to be at all timid about the introduction of the stomach tube; and particularly if there is doubt about diagnosis we should never fail to avail ourselves of it. The most important fact which the investigations of the gastric contents in patients with ulcer has taught us is that there are almost invariably present hyperacidity and hypersecretion of the gastric juice. If the stomach is examined when fasting, we shall find a fluid containing a large amount of hydrochloric acid, and if we examine it an hour after a test breakfast, we shall find an excessively high amount of acid (see page 513). This fact was discovered by Riegel, and we have found it true in all uncomplicated cases of ulcer of the stomach; indeed, it seems to be so much the rule that if anacidity were found it would render any previous suspicion of ulcer extremely improbable. If, however, the ulcer has led to stenosis of the pylorus, and so to extreme stagnation of the gastric contents, there may be temporary subacidity or anacidity (*vide infra*). Likewise—and this is a point of great practical importance—hydrochloric acid

may be absent if a gastric carcinoma has developed on the base of a former gastric ulcer (*vide infra*). Lactic acid is invariably absent when there is hyperacidity from hydrochloric acid; or, at most, if there is great stagnation of the gastric contents, there may be a moderate formation of lactic acid. It is also very important in cases in which there is no vomiting at all, to examine the gastric contents obtained by rinsing out the stomach, with regard to the possible presence of blood. Not only considerable quantities, but particularly also smaller admixtures of blood, which are not recognized macroscopically (the so-called occult gastric hemorrhages), are of great diagnostic importance, as in association with hyperacidity they are strong evidence of gastric ulcer.

The motor efficiency of the stomach in ulcer is at first normal or even somewhat exaggerated, because the digestion of albumen is very energetic owing to the excess of hydrochloric acid in the stomach. But if the ulcer is located in the pyloric region and leads to a cicatricial stenosis, the emptying of the stomach is delayed and a greater or less amount of food will be found seven or eight hours after the test meal. We shall later describe the symptoms of well-marked obstruction of the pylorus. If there is suspicion of an ulcer, the stomach is usually not inflated at all, or if so only by means of the condom (see page 516). If, however, there is a cicatricial stenosis of the pylorus, or an hour-glass contraction of the stomach, our best means of determining the form and size of the organ is by the X-ray examination of the stomach filled with bismuth paste.

There are no other symptoms in gastric ulcer so important as those already described. The appetite in many cases is well maintained, and if the patient takes little food it is only because he dreads the ensuing pain. Eructations may be absent or there may be sour eructations and heartburn, symptoms referable to the existence of hypersecretion and hyperacidity. The tongue is seldom coated, being usually smooth and red; the bowels are apt to be somewhat constipated; the urine is often only very faintly acid, so that there is a tendency to a deposit of phosphates.

The general nutrition of the patient often remains good, but there may be marked emaciation if there is a long-continued diminution in the amount of food taken, or if there is obstinate vomiting. Sometimes, but by no means invariably, the patients display great anæmia. This may precede the ulcer (*vide supra,* ætiology), or it may develop in association with the symptoms of ulcer, and even without any severe hemorrhage to explain it; but that after severe hematemesis there should be a severe anæmia is a matter of course, and has already been alluded to.

An event which has been already mentioned under pathology—namely, perforation due to the ulcer—is of great clinical importance. The two most frequent varieties of perforation are (1) into the peritoneal cavity, causing peritonitis, and (2) into the left pleura or left lung.

Perforation into the peritoneal cavity leads almost invariably to a quickly fatal peritonitis. When the ulcer has previously caused few symptoms, if any, the excruciating abdominal pain, tympanites, vomiting, collapse, and sudden death from peritonitis may abruptly supervene upon a state of apparently perfect health. In other cases in which there has been a previous formation of adhesions, we have an encapsulated abscess, either subphrenic, between the stomach and the diaphragm, or elsewhere in the abdomen. The subphrenic abscess

may contain air as well as pus, and is then termed pyo-pneumothorax subphrenicus. In making a diagnosis of any subphrenic abscess, we should consider the direct and constitutional symptoms, and also the thoracic signs, for the respiration and the inspiratory expansion of the lungs should be normal. If there is perforation outward or into a coil of intestine, recovery may result. More often, however, the termination is fatal, unless surgical intervention is successful.

Perforation into the left pleural cavity we have observed repeatedly. It causes a purulent or septic pleurisy on that side, and pulmonary gangrene may develop at the same time or later, as a result of perforation into the lung. Whenever we meet a case of apparently spontaneous, left-sided empyema, we should at any rate always think of the possibility of gastric ulcer.

A third possibility, which although rare should nevertheless be mentioned, is perforation into the transverse colon with the formation of a gastro-colic fistula. This condition may occasionally be recognized by the alternating discharge of gastric contents from the intestine (slightly altered food remnants and hydrochloric acid) and of fecal intestinal contents from the stomach either through vomiting or through gastric lavage.

In some cases of ulcer we observe signs of threatening perforation—that is, localized pain due to peritonitis, vomiting, and similar symptoms, and yet the case finally clears up. Probably in most such instances there is a limited peritonitis resulting in adhesions. In certain rare instances such chronic peritonitic adhesions in the neighborhood of the stomach are the cause of constant symptoms, especially pain. Surgical measures may be indicated for their relief.

The general course of round ulcer of the stomach varies greatly, as we can see, in different cases. Complete recovery is by no means rare. In other cases the symptoms persist for years with varying intensity. We have already spoken of the hemorrhage and perforation which may suddenly intervene, and of their significance. Relapses are not infrequent, even after apparent recovery. If the ulcer cicatrizes, the scar itself may give rise to persistent disturbances: there may be obstinate cardialgia, or, if the scar is at the pylorus, the symptoms of pyloric stenosis may gradually be developed. This extremely important sequel of gastric ulcer deserves special consideration.

The cicatrization and contraction of a gastric ulcer which result in stenosis of the pylorus may be complete, but it is by no means exceptional for the ulcerative process to persist in other places while there is a scar at the pylorus, so that we may have the symptoms of stenosis combined with all the other symptoms of ulcer, such as pain, hyperacidity, hemorrhage, and perforation. The narrowing of the outlet of the stomach is partly due to the contraction of the scar, and partly to the callous, fibrous thickening of the stomach wall at the edge of the ulcer. Many physicians lay great stress upon a simultaneous pylorospasm—i. e., a tonic cramp-like closure of the pylorus due to irritation of the surface of the ulcer or to hypersecretion. The assumption does not seem to me to be proved. At any rate, in my opinion, the mechanical and anatomical conditions play a much more important rôle than this assumed pyloric cramp. To the mechanical effect of a pyloric stenosis is added sometimes, perhaps, a disturbance of the expulsive power of the stomach in so far as deep-seated ulcers may destroy a part of the muscular coat of the stomach.

The first and most important result of pyloric stenosis is prolonged retention of the gastric contents. So long as the muscular coat of the stomach is able by increased effort to overcome the increased resistance, there is no disturbance to be noticed. It is proper to speak of a compensated pyloric stenosis, just as we speak of a compensated valvular disease of the heart; but if the stenosis becomes greater, so that even the hypertrophied muscular coat ceases to be able to discharge the chyme into the intestine, there is a gradually increasing collection of food in the stomach, and the only way for the organ to relieve itself is by vomiting. The vomiting, in case of pyloric stenosis, is somewhat characteristic. It occurs from time to time, perhaps at intervals of two or three days, and is very profuse. Then, the stomach being partially emptied, new collections may form until their large amount leads to vomiting again. The vomitus in cases of stenosis due to ulcer is usually very acid and contains much free hydrochloric acid, and sometimes blood. The stomach itself becomes gradually and increasingly dilated, because of the stagnation of its contents. Usually this secondary dilatation of the stomach may be recognized by mere external examination. We can feel the stomach as a great limp, flabby bag; and from time to time we can see its walls bulge out from muscular contraction. We can often distinctly recognize the peristaltic waves passing from the fundus to the pylorus. In the region of the pylorus we may sometimes, though by no means invariably, make out a distinct resistance on palpation, suggesting a tumor and corresponding to the thickening of the gastric walls from scar formation. In such cases it is often especially difficult to distinguish between the scar of an ulcer and a carcinoma. Even if we can feel no tumor, visible contractions of the stomach with distinct peristalsis are almost invariably pathognomonic of pyloric stenosis. In such cases there is usually a cramp-like, painful sensation, associated with very marked contraction of the muscular coat. During these contractions of the stomach its contour is often so evident that we can obtain a perfectly distinct idea of its size, extent (often reaching below the navel), and position. But we must especially emphasize the fact that the size of the stomach, its dilatation, is not the essential point. The main thing is the stenosis of the pylorus, and, above all, the degree of consequent motor inefficiency. With regard to the latter, we can reach no complete conclusions without using the stomach tube. With its aid we can determine the amount the stomach contains, and recover ingesta, such as cranberries and green-colored vegetables, which had been eaten many days before. If we empty the stomach completely and give a test meal, we can gauge more accurately the degree of motor insufficiency. We may also determine precisely the position, form, and size of the stomach when emptied, by means of inflation. In exceptional cases there is a large amount of bile mingled with the gastric contents. The author surmises that in such cases the cicatricial process has changed the pylorus into a rigid, though narrow, tube, which can neither be opened nor closed. Thus we have an "insufficiency" associated with the stenosis of the pylorus, so that no obstacle is opposed to the entrance of bile into the stomach.

When there is a well-marked, uncompensated stenosis of the pylorus, there is always a decided impairment of the general health. Nutrition grows more and more imperfect because of the persistent vomiting, and there may be extreme emaciation. Still, many patients with stenosis due to ulcer may

maintain a tolerable condition of health for many years, if they take proper care of themselves by regular washing of the stomach and by eating proper food (*vide infra*); but finally, even in such individuals, the symptoms grow worse, or a new trouble develops, one of which there is always danger in any case of stenosis due to ulcer. We refer to the secondary development of carcinoma at the seat of the former ulcer. This happens not infrequently, and is to be considered at length in the next chapter. There remain to be mentioned some special symptoms, associated with ulcers of the pylorus when they lead to stenosis. There is usually a decided tendency to constipation; the urine is faintly acid and often has an abundant deposit of phosphates; the pulse is often remarkably infrequent; and, finally, there are sometimes attacks of tetany.

Diagnosis.—The diagnosis can be made only when the above-mentioned characteristic symptoms are present. Of these, hematemesis is by far the most significant, for it is with very few exceptions the result of gastric ulcer. Particularly is this true of individuals under middle age.—But how shall we determine whether the blood ejected did not come from the nose or the lungs, rather than the stomach? The answer is not always easy. If an epistaxis occurs at night, a part of the blood often flows back into the nasopharynx, and, being swallowed, excites vomiting, so that a gastric hemorrhage is suggested.

In doubtful cases we must, therefore, make an accurate examination of the nose. It is also very important for the physician to remember that blood may be "vomited" in hysterical cases. If there is also nervous cardialgia, one might easily be misled to assume the existence of a gastric ulcer. Usually, however, we can make sure of the diagnosis by means of the other symptoms of hysteria (see the chapter on Hysteria), and by examining the "vomited" blood. The latter does not come from the stomach at all, but from the gums, the pharynx, or the nasopharynx, and consequently it is almost always comparatively bright-red, rather fluid, and mixed with mucus and saliva; and on microscopic examination it shows pavement epithelium and microörganisms from the mouth.

The diagnosis between gastric and pulmonary hemorrhage in doubtful cases depends on the following factors: 1. The previous condition of the patient—whether he has had cough, expectoration, and other pulmonary symptoms, or, on the other hand, gastric pain and vomiting. 2. On the character of the hemorrhage, whether accompanied by vomiting or by cough. But there may have been both. Violent vomiting may excite a cough; and, on the other hand, blood which has been coughed up may be in part swallowed and induce vomiting. 3. On the character of the blood: if from the lungs, it is usually bright-red and frothy, containing bubbles of air, with few clots, and of alkaline reaction. In gastric hemorrhage it is usually dark, mixed with food, partly clotted, and acid in reaction from admixture with the gastric juice. 4. On the results of physical examination. In this, of course, we must be extremely cautious after a hemorrhage, lest the movements of the patient excite fresh bleeding; and yet we may be able to perceive from the general condition of the patient, or from dullness at the apices, or moist râles, that pulmonary disease is probable. If the blood comes from the stomach, we usually detect nothing but the signs of anæmia. 5. The subsequent symptoms. In cases of pulmonary

hemorrhage there is almost sure to be an expectoration for the next few days, either of pure blood or of matter stained with blood; and, in gastric hemorrhage, the next dejection will almost certainly be black, from the presence of decomposed blood. In doubtful cases the appearance of blood in the stools almost invariably settles the question in favor of gastric hemorrhage. The best way of testing for blood in the feces is by means of guaiac and turpentine (*vide supra*).

If no gastric hemorrhage has ever occurred in the course of the disease, violent and persistent gastralgia of the peculiar character above described is that symptom which would first arouse suspicion of an ulcer, particularly if the pain is from time to time associated with vomiting. If hyperacidity or hypersecretion has been demonstrated, this condition in association with other symptoms may greatly strengthen the diagnosis. Severe gastric pain, combined with hypersecretion, makes the diagnosis of gastric ulcer very probable. To make an absolutely certain distinction between ulcer and nervous hypersecretion (*q. v.*) is impossible, if there is no hematemesis. In general, we must remember that it is often possible to observe more or less distinct "symptoms of ulcer," without being absolutely certain that an ulcer exists. Often we are obliged to await the further course of the disease, and particularly the results of treatment, in order to settle our doubts.

The two diseases which it is most difficult to distinguish from ulcer are nervous dyspepsia with nervous gastralgia, and carcinoma. When we consider these diseases we shall discuss at greater length the differential diagnosis between them and ulcer of the stomach. Cholelithiasis, especially if this has produced chronic cholecystitis and pericholecystitis with adhesions, may also be confused with gastric ulcer. This is true, too, of certain cases of chronic appendicitis. We shall discuss this matter later. Finally, chlorotic patients often have gastric symptoms (see the chapter on Chlorosis) which excite suspicion of an ulcer, especially as hypersecretion of the gastric juice is often associated with chlorosis. Frequently it is impossible to make an absolute diagnosis in these cases, but the results of treatment may be decisive. Preparations of iron, particularly Blaud's pills, are ill borne in ulcer, while in case of genuine chlorosis they usually cause rapid improvement in the gastric symptoms, as well as in the general condition.

Prognosis.—The chief dangers in ulcer of the stomach, hemorrhage and perforation, have already been spoken of. Whether these complications will actually occur in any individual case, and when, we cannot determine.

There can be no doubt that a large number of ulcers heal perfectly; but, as we have already said, even the resulting scar may cause trouble. We must remember the possibility of persistent gastric disturbance, particularly cardialgia, and also the development of a cicatricial stenosis with its consequences. Finally, we have the danger, already mentioned, of the eventual development of carcinoma in the scar of an old ulcer.

Treatment.—If the diagnosis of gastric ulcer is evident, or if the symptoms are of such a nature that there is a justifiable suspicion of an ulcer, the patient should be urgently advised to submit himself to careful and methodical treatment, for it is only by means of a sufficiently persistent and properly conducted treatment that we can hope for good therapeutic results in ulcer of the stomach.

One essential condition is that the patient should keep his bed for the first part of the treatment at least for three or four weeks. Complete bodily rest is certainly important in promoting healing of the ulcer. The patient should also have moist compresses, or, still better, as Leube has advised, warm poultices applied all day long to the epigastrium. These applications are very good for the pain, and they also have a certain psychological importance, for they reconcile the patient to keeping quiet in bed. Thirdly, and this is probably the most important point, the patient must keep strictly to a prescribed diet. This diet we are about to describe at length. The main consideration in determining the patient's diet is that all mechanical and chemical irritation of the floor of the ulcer must be avoided. Fourthly, we should have regard to the almost invariable existence of hyperacidity of the gastric juice. We therefore give to patients alkaline carbonates, either in the form of Carlsbad water (on waking a large glassful of the Mühlbrunnen warmed to about 98.6° F. [37° C.]; then in the forenoon, between ten and eleven, as much more), or, what we consider in many ways still better, bicarbonate of soda (either alone or combined with subnitrate of bismuth). Of the soda we should give as much as is held upon the point of a knife every hour, so as to keep the gastric juice constantly neutralized. With regard to the dietetic treatment of gastric ulcer, the best way is to follow Leube's example, and have four diet lists. Beginning with the first, we gradually go on at definite intervals of time to the second, third, and fourth. For the first ten days of treatment the patient follows the first diet list. This contains boiled milk, broth, unsweetened rusks, softened by soaking; to this we may add various artificial food preparations, such as sanatogen, somatose, hygiama and bioson, and meat juices. Then during the next week we have the second diet list, comprising gruel, soft-boiled rice and oatmeal, eggs, boiled calf's brain and sweetbread, boiled pigeon and chicken. From the eighteenth to the twenty-fourth day of treatment we have, in addition, the third diet, with scrapings of raw beef and beefsteak, potato *purée,* boiled calf's feet, and for beverage some tea or weak coffee. From the twenty-fourth to the thirtieth day the patient has the fourth diet list—that is, broiled fowl or pigeon, partridge, venison, roast beef, tenderloin, finely chopped macaroni, and bread. It is understood that with each advance in diet the previous diet lists are also permitted. In general, the patient has about five meals a day, the amount of each being determined by the appetite and symptoms of the patient. If everything goes favorably, the gastric pain will wholly cease after a few days of this diet, under the other regulations above enumerated. The patient then proceeds toward recovery without any discomfort, except, possibly, hunger. If the transition to a more advanced diet causes pain, we must resort for a few days to the simpler foods. The above suggestions, however, must be regarded as an outline from which certain variations may be made to suit the individual case. Thus, Lenhartz particularly has lately called attention to the fact that some ulcer patients, even after a severe gastric hemorrhage, tolerate a diet of milk, scraped meat, soft-boiled eggs, ham, and butter well, and recover more quickly than under the former stricter *régime.* Such a plan, however, cannot be applied generally. We must ever be guided in each case by the appetite and the symptoms. In some cases the administration of pure olive oil, which may be combined with oil of peppermint, has been very use-

ful. Two and a half to three ounces (gm. 75 to 100) are given daily. The high nutritive value of the oil and its occasional evident analgesic effect are indications for its use. Many patients take the remedy without any aversion. In general, we may be well assured that the results are better and more certain the more strict and methodical the treatment is. If the patient has borne the articles of the fourth diet list for about a week without any discomfort he may go on by slow degrees to other simple dishes, such as veal, pike, trout, porridge, vegetables, and other similar articles, and so to an ordinary diet. Of course caution in eating and drinking is necessary for a long time.

With the mode of treatment just described we shall in most cases accomplish as much as possible; but if, despite this regimen, the symptoms do not cease, there are two other remedies which deserve to be tried—viz., subnitrate of bismuth and nitrate of silver. These are specially employed, also, in treating poor persons whose circumstances often render it impossible to carry out strict dietary rules. The subnitrate of bismuth is almost always given in combination with bicarbonate of soda; of a mixture containing 3 to 5 parts of bismuth and 30 parts of soda and 5 of the oleosaccharate of peppermint, we give several times a day a generous "knife-point full" before meals. In severe and obstinate cases of ulcer, we can from our own experience recommend also "injections" of bismuth (Fleiner). The patient's stomach is washed out in the morning before breakfast, and then ʒ iv to v (gm. 15 to 20) of the subnitrate of bismuth suspended in a glass of warm water is poured slowly into the stomach tube; the patient should at the same time lie on his right side, and so remain for at least half an hour. The heavy bismuth powder is expected to deposit itself upon the surface of the ulcer (which is probably in the pyloric portion of the stomach), and thus in a mechanical way protect it from irritation and promote its healing. The symptomatic results of this method are sometimes excellent. Often, also, favorable results are obtained from nitrate of silver. This may be partly explained by its neutralization of the hydrochloric acid and the formation of chlorid of silver. The drug is prescribed in a solution of 6 gr. to 4 ounces of water (gm. 0.3 to 120), to be kept in opaque glass, and of this a teaspoonful should be given three or four times a day before meals.

If, in spite of a continuous, methodical, medical treatment, we obtain no marked result, then there usually exist special anatomical conditions, such as cicatricial stenoses and deformities of the stomach, hour-glass stomach, or chronic adhesions with neighboring organs, etc., which cannot be influenced by the treatment. In such cases we are justified, provided the symptoms are severe and continuous, to advise surgical intervention (gastro-enterostomy, separation of adhesions, extirpation of the cicatrix, etc.).

Sometimes the violent pain does not abate under the ordinary treatment, and demands symptomatic remedies. Of course, morphin is the most efficient, given internally or subcutaneously. Chloroform may also be tried: we give a tablespoonful of a mixture containing 1 part to 120 of water. I have occasionally seen excellent results from orthoform, 15 gr. (gm. 1.0) several times a day, and more frequently from anæsthesin, 5 to 10 gr. (gm. 0.3 to 0.5) in powder form, several times daily. Gerhardt recommends for the gastric pain 3 or 4 drops of the solution of perchlorid of iron in a wineglass of water.

Excessive vomiting and persistent nausea are likewise to be combated by

the narcotics. Opium is the best; morphin, cocain, chloral, and bromid of potassium may also be tried. At the first appearance of blood in the vomitus the greatest bodily quiet and most careful dieting is absolutely indispensable. For the first day or two it is best to allow nothing except ice-cold milk and bits of ice in the mouth to appease the burning thirst. In severe cases we may try giving water and milk by the rectum, so as to afford the stomach complete rest. The patient must lie as quietly as he can. A flat ice bag, not too heavy, should be placed on the epigastrium. In case of persistent nausea or eructations, small doses of opium or morphin are to be prescribed. If the hemorrhage is obstinate we may try the subcutaneous injection of ergotin or cornutin, or, by mouth, acetate of lead or chlorid of iron. Gelatin injections (see page 33) may also be tried. In severe cases, finally, surgical measures (gastro-enterostomy, jejunostomy) have repeatedly been tried.

We must wait four or five days after a hemorrhage before we can give a little more food by the mouth, and this must be allowed cautiously and in a liquid form. [If in persistent hemorrhage or obstinate irritability of the stomach we wish to give the organ complete rest, we may try to supply food by rectal injection and moisture by normal saline infusions under the skin, 1 or 2 pints twice a day. Morphin ($\frac{1}{24}$ gr.) may be given every four hours to lessen hunger.]

If peritonitis appears as the result of perforation, the best means to try are the outward application of ice to the epigastrium and the internal use of opium in large amounts—that is, half a grain to a grain (gm. 0.03 to 0.05) every two or three hours, or 20 to 30 drops of laudanum. Unfortunately, however, the cases are exceptional in which the peritonitis does not become general. The only remaining hope lies in surgical interference, although the results even of laparotomy are dubious. The earlier the laparotomy is performed in cases of perforation, the better are the chances of success.

If the symptoms of pyloric stenosis are growing more and more distinct—that is, if the hypertrophied muscular coat of the stomach is no longer vigorous enough to prevent the stagnation of food, with consequent dilatation of the stomach and vomiting, we can perhaps restore the patient to a comfortable condition by means of regular washing out of the stomach, combined with a suitable diet. This irrigation prevents any great and burdensome accumulation of food in the stomach. Moreover, it rids the patient, for a time at least, of the large amount of strongly acid gastric juice which is usually present. These beneficial and agreeable effects are soon so noticeable that often the patient learns to rinse out his stomach himself, or even, by bending over and pressing, to empty out the gastric contents through the tube. As a rule, in cases of gastrectasia resulting from pyloric stenosis, we wash out the stomach every morning before breakfast. Ordinary lukewarm water suffices, but if there is an excessive secretion of hydrochloric acid it is satisfactory to employ one- to two-per-cent solutions of bicarbonate or borate of sodium. In severe cases the operation may be repeated in the evening before supper.

The diet in stenosis following ulcer should consist of such articles of food only as can pass with comparative ease through the narrow pylorus, being at the same time highly nutritious. Most suitable are milk, eggs, soups with the addition of meat juice, somatose, oatmeal, rice, or flour; gruel made from rice, oatmeal, or preparations like Mellin's food; and, finally, finely chopped

meat of all sorts. The meals should not be too large, but should rather be frequently repeated—a little food often.

In this way it is often possible to give the patient considerable relief for many years, and even to make him gain weight. Other methods of treatment —by applying electricity to the stomach, by cautious massage of the organ, or by the administration of strychnin—may be employed now and then in practice, but they have no special value in actual stenosis.

It may well be questioned whether the existence of such patients, who are preserved from excessive discomfort only by means of daily lavage and a monotonous and limited diet, is really bearable; and, still further, what are we to do when, in spite of the most skillful treatment by the method described, the symptoms persist and the nutrition and strength of the patient grow gradually more and more impaired? For such there is still the possibility of actual and complete recovery. We refer to surgical intervention. In no other severe disease of the stomach do operative procedures offer so great a prospect of favorable result as in stenosis of the pylorus following ulcer, and therefore it has been our rule for a series of years, in every severe case of this sort, to urge the patient strongly to an operation. Moreover, we should not delay too long, if the patient is already enfeebled, or if there is reason to suspect that carcinoma is developing. The operation is usually gastro-enterostomy, or more rarely pyloroplasty; but we cannot enter here into particulars. Certainly we ought not to conceal from the patient that we have to do with a serious operation, which is not absolutely without danger; yet in the great majority of cases the results of treatment are excellent. After a successful operation the patient is freed from all discomfort, he regains the appearance of health, and sometimes even the dilatation of the stomach completely disappears. It is true that later, perhaps years later, relapses may occur, whether because of special mechanical conditions or from the development of secondary carcinoma; but even then perhaps assistance will be given by a second operation.

CHAPTER VI

CANCER OF THE STOMACH

Ætiology.—We cannot here discuss the ætiology of carcinoma in general, and we shall therefore merely enumerate the factors which experience has shown to favor the development of cancer in the stomach.

Age has a remarkable influence. Gastric cancer is decidedly most frequent late in life, between the fortieth and sixtieth year. Still, it is not very rarely seen in younger persons. We have ourselves seen several cases in persons between twenty-two and twenty-five years of age.

Sex is of no importance.

Heredity has a slight but undeniable influence. The more closely we search, the more frequently shall we be able to demonstrate an hereditary predisposition to cancer. The most famous example of the transmission of cancer is presented by the family of Napoleon.

The relations of gastric cancer to previous disease of the stomach is very

important. It is not probable that the tendency to cancer is increased by frequent errors in diet, indulgence in alcohol, or similar causes. Most of those who are attacked by gastric cancer have previously lived in a perfectly temperate manner, and report that they have thus far always enjoyed a good digestion. The exception is to discover that there has been any special injury or trauma, such as scalding or corrosive poisoning, and if there is a history of them they are to be regarded as merely contributory causes. Far more important, however, is the fact which the investigations of Hauser have lately confirmed, that gastric carcinoma is not infrequently developed in the cicatrized edges of a previous gastric ulcer. We have ourselves had a considerable number of cases in which both the clinical course of the disease and the results of the autopsy illustrated this mode of development. We shall later discuss in greater detail certain important clinical peculiarities of this "ulcer carcinoma."

Pathology.—The stomach is a favorite seat for cancer. About a third of all cases of cancer are gastric. The parts of the organ most often attacked are the pyloric end and the lesser curvature. Less frequently the cardiac end and the fundus suffer.

The new growth takes the form either of a circumscribed tumor or of a diffuse infiltration, thickening the walls. The disease invariably originates in the mucous layer, extending thence after perforating the muscularis mucosæ into the submucous, muscular, and serous coats. Often the cancer extends by way of the lymph-channels, or sometimes the veins. The lymph-glands and the omentum are frequently attacked by the new growth. Moreover, we find metastases in the peritoneum, and also with particular frequency and abundance in the liver, whither cancerous cells are carried by the branches of the portal vein. The connective and muscular tissues in the neighborhood of the cancer are quite often considerably hypertrophied.

Histologically, gastric cancer is of the cylindrical-cell variety, starting from the glandular epithelium. The soft tumors are termed medullary; the firm and hard, scirrhous or fibroid. The medullary cancers are particularly apt to be quite extensively broken down on their exposed internal surface, thus forming what are known as cancerous ulcers. This seems to be mainly the result of the gastric juice acting on the superficial and insufficiently vascularized portion of the tumor. The base of these ulcers is therefore usually clean. Not infrequently, however, the base has a necrotic and gangrenous character and parts of the tumor may slough off. In many cases affecting rather young subjects, and sometimes in others, we find colloid cancer. This form also may appear either in nodules or as a diffuse growth, infiltrating the tissues. It is peculiarly apt to involve the peritoneum extensively, with consequent ascites.

Clinical History.—Most cases of gastric cancer exhibit a combination of grave digestive disturbances with a relatively rapid loss of flesh and strength. Now and then the gastric symptoms assume less prominence. The chief sign of disease is a constantly progressive marasmus or anæmia, the true cause of which is either entirely latent or not unmistakably recognizable till late in the illness.

Some of the gastric symptoms are not very characteristic. They merely show that digestion is disordered. The tongue is usually thickly coated and

dry. There is loss of appetite, often a special aversion to meat, and distress after meals. The patient complains of a disagreeable sensation of pressure in the epigastrium, increased by food. Sometimes this amounts to actual cardialgia. A constant painful sensation in the gastric region and also burning pains in the back are frequently observed. Some patients are annoyed by eructations. Occasionally vomiting is troublesome; in other instances there is scarcely any. The constant recurrence of vomiting is an almost sure sign that the cancer is situated in the pyloric region and obstructs the orifice. The vomitus may contain nothing but mucus and ingesta; or it may assume, from the admixture of blood, a very characteristic and somewhat pathognomonic appearance.

Free gastric hemorrhage and consequent hematemesis is exceptional, or at least it is much less frequent than in ulcer of the stomach; but the vomitus often contains decomposed blood, and in many cases this will be for a time almost a constant appearance. Most of the ulcerated cancers bleed frequently, a little at a time. When it reaches the stomach the blood is decomposed and gives that well-known "coffee-grounds" or "chocolate-colored" appearance to the vomitus, or to the gastric contents obtained by means of a stomach tube, which is so important a symptom for the diagnosis of cancer of the stomach. In such cases the best means for demonstrating the presence of blood, in the brownish or blackish fluid, is the guaiac-turpentine test (*vide supra,* page 517). This gives an extremely distinct reaction in the case of carcinoma of the stomach, because lactic acid also is usually present. We should mention that in the case of ulcerating carcinoma ventriculi the vomitus may have so foul an odor that it may even be regarded as stercoraceous.

Immediate microscopic examination of the vomitus may reveal red blood globules. Further, the vomitus consists of partially digested food, and also contains an abundance of microörganisms. Sarcinæ are found much less often in carcinoma than in ulcer of the stomach, but we frequently see under the microscope an abundance of yeast cells, and of long threadlike bacilli which have a relation to lactic-acid fermentation (*vide infra*). It is extremely rare to find in the vomitus characteristic particles of cancer. It is somewhat less unusual to find little pieces of the growth attached to the stomach tube when the carcinoma is at the cardiac end or in the fundus of the stomach. These, if obtained, are, of course, to be submitted to microscopic examination.

External objective examination. Sometimes we can clearly recognize by mere inspection the position and size of the organ, any peristaltic motions (*vide infra*) that there may be, and even in some cases the presence of a tumor.

Palpation must be practiced with especial care in every doubtful case. It is carried out with the patient in a completely horizontal dorsal position, or occasionally, under special circumstances, in a lateral position. We must try to obtain the greatest possible relaxation, and, if necessary, we should palpate while the patient is in a warm bath. We palpate alternately with gentle and with deep respiration to determine the mobility of any existing tumor. It is often useful to exert counter pressure on the soft parts by placing the left hand under the patient's back. We must always begin with very gentle palpation and then gradually use deeper pressure. In a great many cases the new growth can be felt with distinctness through the abdominal wall as a hard irregular tumor. It is often useful to hold the fingers quiet and press lightly

on the abdominal wall while the patient breathes deeply. We then occasionally feel the tumor gliding downward under the fingers. The situation of the tumor is, in a majority of cases, the epigastrium. Carcinoma of the pylorus, which is the most common, is usually felt in the middle or in the right half of the epigastrium; carcinoma of the greater curvature somewhere in the level of the navel. But it should be considered that the position of the stomach may be considerably altered by the presence of a growth—for example, we have seen a case of pyloric cancer with secondary dilatation of the stomach, in which the pylorus had sunk so low that the tumor could be felt about a hand's breadth above the symphysis. In some cases the tumor varies its position (*vide infra*) according to the degree of distention of the stomach. The influence of respiration upon the tumor is various. The majority of gastric cancers, particularly cancers of the lesser and greater curvatures, move with respiration. When displaced downward by inspiration they may be held in that position with the hand, returning to their ordinary location when the hand is removed. Tumors of the liver and cancers of the stomach which are adherent to the liver cannot be thus held down. When the carcinoma is situated high up, in the lesser curvature or near the œsophagus, it may not be felt at all except during inspiration.

In a minority of the cases no tumor can be felt at any time. The tumor is undiscoverable, first, in most cases of diffuse cancerous infiltration of the stomach walls. We may, indeed, notice an increased sense of resistance and hardness in the epigastrium, but we cannot refer this condition with certainty to a new growth. Secondly, the new growth may extend chiefly inward, toward the cavity of the organ, and may thus escape detection. And, finally, the tumor may be so concealed by the liver or the edge of the ribs that it is inaccessible to the touch. Such cancers as attack the cardiac extremity, the posterior wall, or the lesser curvature of the stomach, are particularly apt to be out of reach of palpation.

Percussion of the cancer rarely gives flatness, but instead a muffled tympanitic resonance. This is sometimes an influential factor in the differential diagnosis from cancer of the liver. In a few cases, upon auscultation in the neighborhood of the tumor, we may hear a soft systolic murmur, probably due to compression of a rather large artery.

Examination of the gastric contents. While introducing the stomach tube, or perhaps, if need be, a less flexible instrument, we should notice whether the tube enters the stomach with ease. Carcinoma at the cardia is not infrequently the cause of a sensible resistance to the tube when it passes the lower end of the œsophagus. With regard to the examination of the gastric contents, the chief point is about free hydrochloric acid. Von der Velden first pointed out what numerous investigations have since confirmed, that in almost all cases of gastric carcinoma there is no free hydrochloric acid, so that the phloroglucin-vanillin reaction an hour after the test breakfast is negative. Associated with other symptoms this is an extremely important diagnostic sign (*vide infra*). The cause of this almost invariable absence of free hydrochloric acid in carcinoma of the stomach has not yet been determined, especially as this symptom has often been observed very early in the course of the disease. The associated catarrh of the gastric mucous membrane and its secondary atrophy may have some influence, but probably there are also other unknown

causes at work. An examination for pepsin may be omitted in practice; it also often turns out negative. Far greater importance attaches to the fact that the gastric contents in carcinoma are very apt to show a surprising amount of lactic acid. This is almost invariably so when the carcinoma obstructs the pylorus. The explanation of this is very simple. There is no hydrochloric acid to exert an antiseptic action, and the food is stagnant in the stomach, so that there is an especially favorable opportunity for the development of a vigorous lactic-acid fermentation. There is, then, nothing specific about the presence of lactic acid in the gastric contents in cases of carcinoma, only it is a very frequent and therefore important symptom.

The producers of lactic-acid fermentation in the stomach contents are the lactic-acid bacilli [Oppler-Boas bacilli] which are usually readily demonstrated microscopically whenever the gastric contents contains lactic acid. They are distinguished by their growth into long threads. The microscopic demonstration in the gastric juice of the "long bacilli" (*vide* Fig. 72), which may also be easily grown in pure culture, has therefore no slight diagnostic significance in the recognition of carcinoma of the stomach.

The motor efficiency of the stomach is impaired in most cases of carcinoma, except in cancer affecting the cardia, or in small cancers on the fundus. Whenever there is an extensive development of carcinoma of the stomach, the muscular coat of the organ is sufficiently damaged to embarrass its motor activity; but of course the hindrance to the discharge of its contents is greatest when the new growth is at the pylorus. In this case, besides the direct destruction of the muscular tissue, we have the purely mechanical stenosis, and this may at last become extreme. For this reason, if we find a marked stagnation of the ingesta we can usually infer that the seat of the carcinoma is the pylorus (*vide infra*). We should not omit to inflate the stomach when it is possible. We can thus determine not only the size and position of the stomach, but also its relations to any tumor that may be felt; and finally, it should be added that a test for blood (*vide infra*) should be made with the fluid obtained by washing out the stomach.

Certain peculiarities in the clinical picture of gastric carcinoma are occasioned by its special location; if at the cardia, the symptoms are similar to those of cancer of the œsophagus. As soon as the cardia is narrowed, the patient himself feels that it is difficult for food to enter the stomach, and speedily there appears vomiting, or, more accurately, regurgitation of food, so that the patient can take nothing but liquid nourishment. The diagnosis of carcinoma at the cardia is usually easy. We feel the resistance at the cardia with the stomach tube some 16 to 18 inches (40 to 45 cm.) from the front teeth. Externally the growth cannot be felt until it invades the lesser curvature of the stomach. Then we often feel the tumor in the epigastric angle, especially on deep inspiration. In carcinoma of the fundus the symptoms may be indefinite for a long time. The pylorus remaining permeable, there may be no vomiting at all. In late stages the tumor which may be felt is often very large, particularly if there is involvement of the omentum, which changes it into a great cancer mass.

When cancer at the pylorus leads to stenosis, the clinical picture is especially important as well as very common and very characteristic. As soon as there is a hindrance to the emptying of the stomach, whether because the muscular coat is impaired by infiltration with carcinoma or because there is the direct mechan-

ical obstacle of stenosis, there result retention and accumulation of food in the stomach, with exaggerated peristalsis, and finally gastric dilatation. The circumstances are precisely similar to those depicted in the preceding chapter when speaking of cicatricial stenosis (*cf.* page 536). In advanced cases the contours of the dilated organ are clearly seen through the emaciated and collapsed abdominal walls. From time to time a contraction occurs, and we see the peristaltic wave slowly travel toward the pylorus. Very often the carcinomatous growth near the pylorus is distinctly palpable, and even visible. Often it moves with respiration. Washing out the stomach will demonstrate the stagnation of its contents, and by inflation we determine its size and position. If the stomach is not emptied artificially, from time to time very large amounts are vomited, the vomitus being invariably devoid of hydrochloric acid but containing much lactic acid, and sometimes also showing fermentation (*vide supra,* page 510).

In addition to the symptoms of gastric cancer, referable to the stomach itself, the greatest attention should also be paid to the general disturbance of nutrition occasioned by this gastric disease. Loss of flesh is not rarely the very first symptom which calls the patient's attention to his disease. This wasting is observed earliest in cases which are attended with anorexia and vomiting. The patient also gradually takes on that familiar sallow cachectic look which is characteristic of most cases of cancer. This abnormal color of the face is all the more striking because patients with gastric cancer comparatively rarely have marked gray hair. As my teacher, Wunderlich, always emphasized in his clinic, and as I can confirm from my own experience, it is certainly remarkable how frequently even elderly patients with gastric cancer (over fifty years of age) have hair that is scarcely tinged with gray. Some patients become excessively anæmic. The skin acquires a waxy pallor, and there are all the symptoms which result from great anæmia, such as cerebral disturbances and functional cardiac murmurs. Sometimes the blood itself presents decided peculiarities in such cases. Thus we may find microcytes and poikilocytes in it. Gastric cancer and pernicious anæmia (*q. v.*) have been repeatedly confounded. In one such case we made the interesting discovery of extremely abundant metastatic cancer in the bones. As the bone marrow is known to have something to do with the production of the blood, it may be that the anæmia was due to this abnormal condition. At any rate, the grave anæmia which results from cancer cannot be regarded in just the same light as the loss of flesh and the cachexia. We often find extreme anæmia in patients who are tolerably well nourished, while, on the other hand, many patients with carcinoma, though emaciated to a skeleton, do not display this peculiar anæmic pallor. The anæmia must, therefore, depend upon some special circumstances, and I have no doubt that it is most frequently caused by continued though small hemorrhages, due to ulceration of the new growth. In other cases, however, it would seem that the blood is also affected by specific harmful influences.

Special derangements of other organs are relatively infrequent. Metastatic cancer is of importance. It attacks the liver chiefly. If the hepatic new growth is considerable, it may quite overshadow the primary cancer—there being jaundice and a greatly enlarged, nodular, and tender liver. Secondary carcinosis of the peritoneum is also apt to cause marked symptoms, such as ascites and abdominal pain. Extensive carcinoma of the omentum often produces large

tumors which may be felt even during life, and which are difficult to differentiate from the actual stomach tumor. Large tumors, lying transversely across the epigastrium, are frequently found at autopsy to be a carcinomatous degenerated and shrunken omentum. The fact is very important that we not infrequently can demonstrate peritoneal metastases in the true pelvis by rectal or vaginal examination. Secondary cancer may also involve the mesenteric and retroperitoneal lymph-glands, the lungs, and other organs, but it does not usually give rise to striking symptoms when so situated, although the discovery of even small metastases by palpation may have great diagnostic significance. Metastatic lymph-gland enlargement in the left supraclavicular fossa (so-called Virchow's gland) is not common, though I have repeatedly observed it in characteristic form. Swelling of the inguinal glands is also important. We have seen small metastases in them several times. Finally, we would like to call special attention to the appearance of small secondary nodules at the navel, a fact which I have repeatedly noticed. The discovery of such a growth is of great diagnostic value when no primary tumor can be made out by palpation. Occasionally a rounded, hard strand can be felt running from the umbilicus downward along the linea alba—a carcinomatous lymphatic vessel(?).

Direct extension of the new growth into neighboring organs is not very frequent. We will venture to mention one case which we saw, on account of its great rarity. The new growth caused adhesion of the anterior wall of the stomach to the abdominal walls, and then, penetrating through them and the skin of the epigastrium, finally appeared as a tumor, of about the size of one's first, projecting outward. If a cancer ulcerates, it may destroy all the layers of the stomach, and result in perforation and secondary peritonitis; or, if previous adhesions have been formed, the perforation may open up an abnormal communication between the stomach and some neighboring part of the intestine. The transverse colon is the part usually perforated; less often the small intestine. With perforation into the colon a temporary communication may form between this organ and the stomach. Then, as we have had occasion to observe in one case, feculent masses may be vomited, and the stool present the appearance of gastric contents.

As to the bowels, constipation is the rule. Diarrhea is rare. A dark appearance of the stool due to admixture of blood may be of diagnostic significance. The urine is usually pale and but slightly acid. Its amount is diminished, as we should expect from the slight amount of nourishment taken, and from the vomiting. Over the heart we may sometimes hear soft anæmic murmurs. The pulse is usually accelerated, although, if there be extreme marasmus, it may be slow. The blood shows the microscopic changes of anæmia (*vide supra*) and a slight leucocytosis, which latter fact may, under certain conditions, be used in the differential diagnosis from pernicious anæmia.

The temperature is often normal. If the patient is greatly emaciated it is not infrequently subnormal, but, on the other hand, if the thermometer is carefully used we shall very often find occasional irregular elevations of temperature, 100.5° to 102° F. (38° to 39° C.), or even a persistent or intermittent fever. The cause of these elevations, exclusive of complications, is probably, in most cases, the absorption of septic material from the ulcerated surface of the carcinoma. If there are hemorrhages, another factor may be the absorption of the decomposed blood (fibrin ferment). Not infrequently, in advanced stages

of the disease there is more or less œdema of the ankles, hands, and other parts. The explanation of this is the same as in most cases of œdema in cachectic and anæmic patients—viz., the impaired nutrition of the vascular walls, the hydremia, and the associated cardiac weakness. Sometimes there also occur marked pains in the arms and legs. The temperature is normal, or even subnormal. If there is some inflammatory complication, or if the anæmia is extreme, fever may occur.

The entire duration of the disease may be one or two years. It is exceptional for it to last longer, except when the cancer develops in the floor of a preexisting ulcer. In this case the symptoms of gastric ulcer pass into those of carcinoma. The change may be gradual, but there may be an intervening period of apparent health. We have by careful questioning repeatedly been able to make the diagnosis of this transformation during the life of the patient, in cases later confirmed by autopsy. In individual instances the disease, of course, exhibits many variations and departures from the typical course. Sometimes the constitutional symptoms of weakness and emaciation are more prominent, and sometimes the distinctively gastric disturbances.

The fatal termination is usually preceded by the symptoms of constantly increasing weakness. It may be hastened by complications, such as perforative peritonitis. Now and then grave nervous symptoms appear, often quite suddenly. The patient falls into a condition resembling that of diabetic coma (*q. v.*), he is somnolent, and has a peculiar dyspnœa, with deep and labored respirations. Such an attack is probably due to autointoxication, and almost always ends fatally. Recovery from cancer of the stomach is unknown.

Diagnosis.—Gastric cancer is a comparatively frequent disease, and in every case in which there are well-marked gastric symptoms, particularly in an elderly person, the physician should think of the possibility of this grave disease. The suspicion is all the more justified when the symptoms occur in a previously healthy individual without known cause, and if they are associated from the start with a cachectic look and a feeling of weakness and languor, as well as with marked emaciation. If now, having such suspicions, we wish to reach a diagnosis, the first requirement is a thorough investigation of the stomach. External examination should be particularly directed to the discovery of a palpable tumor, if any exists. We should examine while the patient is lying on his back or on his side. If possible, the stomach should be empty and we should palpate while the patient is breathing quietly, and also while he is taking deep inspirations. If a tumor can be felt, the next question is whether it actually arises from the stomach or from some other organ. In most cases the other clinical symptoms, such as vomiting, have already indicated the stomach as the seat of the disease, and thus we know the origin of the tumor. On the other hand, however, there may be great obscurity, and many cases of gastric tumor have been confounded with carcinoma of the left lobe of the liver, the pancreas, the omentum, the transverse colon, and other parts. It is difficult to lay down special rules for diagnosis, because the difficult cases almost invariably have their own special peculiarities. Of importance in every case are an accurate local examination, a mapping out of the neighboring organs, inflation of the stomach and perhaps also of the colon, determining whether the mass moves with respiration, and a consideration of all the other factors. Among these last the most important is the result of an examination with the stomach tube. The

main question in every case is the presence or absence of free hydrochloric acid in the gastric juice. If it is absent on repeated examination our suspicion of carcinoma is confirmed, provided the other symptoms point in the same direction; but if they do not, the absence of free hydrochloric acid is by no means so grave a matter, for it is absent often enough under other circumstances. It is, however, a fact of great practical importance that when free hydrochloric acid has been distinctly and repeatedly demonstrated in the gastric juice, gastric cancer is quite unlikely, even if other symptoms point to it.

In those cases alone, where a carcinoma has developed on the basis of an old gastric ulcer, the gastric contents may, at least in the beginning, still show a greater or lesser amount of free hydrochloric acid. It is often impossible to make the diagnosis of a cancer just developing when it is certain that there is pyloric stenosis due to ulcer. If there is no free hydrochloric acid, but lactic acid, and also impaired motor efficiency, the circumstances are different. The diagnosis of pyloric cancer is then almost certain. The demonstration of impaired motor efficiency is therefore at least as important as the examination for free hydrochloric acid. In case of stasis with simultaneous absence of hydrochloric acid, even if no tumor can be felt, there is great probability of a gastric carcinoma, probably involving the pylorus.

Very important, also, are the other characteristics of the gastric contents, the defective digestion of food, the putrid odor, and above all the demonstration of blood in the gastric contents (*vide supra,* page 517). If we have distinct "coffee-ground" vomitus, and at the same time achlorhydria, these factors alone almost establish the diagnosis, but even smaller amounts of blood, only chemically demonstrable, are important. Finally, we should bear in mind that when no gastric tumor is made out there may be metastatic growths which can be felt—for example, in the lymph-glands—and thus a diagnosis of cancer confirmed.

FIG. 72.—Gastric Contents in Cancer of the Pylorus. *a.* Long (Oppler-Boas) bacilli. *b.* Yeast cells. *c.* Fat droplet. *d.* Fatty-acid crystals. *e.* Starch granules. *f.* Detritus. *g.* Leucocyte. *h.* Erythrocytes.

In case of symptoms which indicate stenosis of the pylorus, with subsequent gastrectasia, it is of great practical importance to determine whether they are due to cancer or to the scar of an ulcer. In this instance, even if we feel a tumor, that fact is not decisive, for the cicatricial hyperplasia of gastric ulcer may cause a distinct tumor, although a comparatively small one; but, as a rule, it is not difficult to reach a decision. Suggesting a scar from ulcer would be: long duration of the disease (two years, sometimes four or five years, and longer); youth of the patient; comparatively healthy appearance; distinct history of ulcer; cardialgia; profuse hematemesis; and finally,

as a decisive factor, the demonstration of an abundance of hydrochloric acid and hypersecretion in the stomach. Under these circumstances the differentiation of stenosis from ulcer is absolutely certain. On the other hand, there is complete certainty of a cancerous stenosis if the disease has developed in a comparatively brief time—in one or two years; if it has attacked an elderly person; if there is distinct cachexia; and, most important of all, if hydrochloric acid is never demonstrable in the gastric contents, while there is an abundance of lactic acid. Other cases occur which are not typical nor of easy diagnosis. Age, duration of the disease, and previous symptoms may suggest a stenosis due to ulcer, and yet on washing out the stomach we find no hydrochloric acid at all, or, at most a faint trace. In these cases we are usually justified in assuming that carcinoma has developed secondarily on the floor of a former ulcer. It must be confessed that in simple stenosis following ulcer, when there is an excessive stagnation of the gastric contents, there may be temporarily a complete combination of all the hydrochloric acid with albumen, and consequently no reaction for free hydrochloric acid; but if the stomach is washed out a few times we soon find an abundance of free hydrochloric acid. On the other hand, when a stenosis due to ulcer has lasted for a long time, our suspicions should be aroused if the reaction for hydrochloric acid is persistently feeble or at times absent, and in its place or along with it lactic acid appears in the gastric contents. We have ourselves repeatedly seen this gradual transition of simple stenosis, due to ulcer with hypersecretion, into carcinomatous stenosis with achlorhydria. Finally, it should be remarked that there is also a stenosis of the pylorus due to simple hypertrophy of its walls, without any ulcer or carcinoma. We have ourselves seen a few cases of this sort which presented the ordinary symptoms of pyloric stenosis with great dilatation, and ended fatally. The pathogenesis of this rare condition has not yet been explained. We venture to suggest that perhaps it may be due to congenital anomalies, more especially as this type of pyloric stenosis has also been repeatedly observed even in suckling infants. Of late, these cases have also been frequently termed pylorospasm, though the assumption of an habitual cramp-like condition at the pylorus is still purely hypothetical. As yet, unfortunately, no particulars are known as to the constituents of the gastric juice in these cases, and for the present it is scarcely possible to make a diagnosis of them with certainty, except in pyloric stenosis of infants.

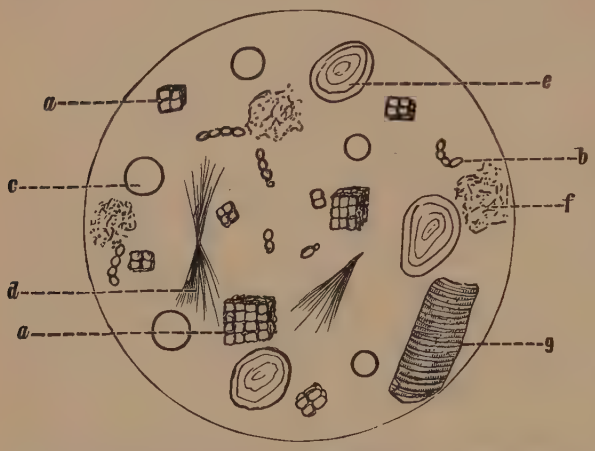


FIG. 73.—Gastric contents in benign stenosis. *a*. Sarcinæ. *b*. Yeast cells. *c*. Fat droplet. *d*. Fatty acid crystals. *e*. Starch granules. *f*. Detritus. *g*. Muscle fiber.

Treatment.—If once the diagnosis of gastric carcinoma has been settled, the question arises whether there is any definite prospect of success from surgical treatment. If not, our treatment can be merely symptomatic with the object of alleviating the patient's suffering, and preserving his strength as long as possible.

The first point in symptomatic treatment is a regulation of the diet. In general, we advise mainly liquids and soft foods, including milk, gruel, rice, sago, delicate vegetables, potato *purée,* softened zwieback, toast, or crackers, and delicate puddings. We may also permit finely chopped or scraped meat, or, still better, fowl; and fish, such as trout, pike, and perch-pike. Such artificial foods as somatose, neutrose, beef peptone and beef juice, and infants' foods, do good service for a time. If the appetite fails we order stomachics—for example, compound tincture of cinchona. The fluid extract of condurango deserves a special mention, and has been a favorite prescription in gastric carcinoma since condurango bark was recommended by Friedreich. We usually prescribe hydrochloric acid after meals (15 to 20 drops of the dilute acid in water) in the hope of making up for the achlorhydria. Pain is alleviated by narcotics, such as morphin, opium, codein, belladonna, chloroform, and anæsthesin, and by moist warm or cold applications. Warm poultices on the epigastrium for several hours a day are usually beneficial. In some cases I have noticed a striking analgesic effect from repeated exposure of the gastric region to the Röntgen ray.

To combat obstinate vomiting we may give the narcotics just named or bits of ice, but the best remedy is rinsing out the stomach, provided the patient is not too feeble. If there are sour or offensive eructations, we prescribe bicarbonate of soda, magnesia, or powdered charcoal. If the pyloric cancer is leading to stenosis with stagnation of food in the stomach, the daily washing out of that organ is an excellent means of relieving the patient for a time, at least, of a great part of his discomfort. If this is practiced regularly with either pure water or a one- or two-per-cent solution of hydrochloric acid, and if the diet is suitable, there may be a great temporary improvement in the condition and even a not inconsiderable gain in weight. But, despite all our efforts, the symptoms may become worse and worse till, finally, the subcutaneous injection of morphin may be indispensable. Then our whole object is to alleviate the patient's sufferings, and to give him moral support and encouragement.

When the vigor of the patient is such as to permit the consideration of surgical interference, the more promptly the operation is undertaken the more chance is there of eradicating the disease and obtaining permanent recovery. It is, therefore, permissible when there is incipient disease and the diagnosis is not yet fully established, although somewhat probable, to propose to the patient an exploratory laparotomy, to be immediately followed by a regular operation, provided the case turns out to be a suitable one. The results of operations on carcinoma of the stomach have not been very brilliant. Still, in many cases the surgeon is fully justified in endeavoring to combat an otherwise absolutely hopeless disease, since there is some prospect of recovery. Although there have been many failures, surgeons have obtained a number of extremely satisfactory recoveries under these circumstances. If there is a stenosis of the pylorus and yet it is no longer possible, or at least no longer

wise, to extirpate the tumor, gastro-enterostomy may give very great relief. If the patient survives the operation, the suffering is much diminished and nutrition may decidedly improve. Many cancer patients enjoy a comparatively comfortable existence for one to one and a half years after a successful gastro-enterostomy. For the particulars we refer to surgical treatises.

CHAPTER VII

ANOMALIES OF THE SECRETION OF GASTRIC JUICE

(Achylia Gastrica and Hypersecretion of the Gastric Juice)

In the present chapter are to be considered those dyspeptic conditions which are associated with a change—that is, either a diminution or an increase—in the secretion of the gastric juice, independently, at least in most cases, of any definite anatomical lesion. We have here to consider, therefore, no marked catarrhal condition or new growth of the stomach, nor gastric ulcer with hypersecretion. The cause of the change in secretion in the cases here considered must rather be sought in the disturbances of the activity of the cells, the special occasion of which is as yet almost entirely unknown. Many clinical observations, it is true, indicate that abnormal nervous influences are important in this connection, but, on the other hand, it is possible that the explanation lies in primary changes in the minute structure and chemistry of the secreting cells themselves. Besides this uncertainty of ætiology, there is also an absolute lack of thorough anatomical investigations in this regard, so that it is at present a very difficult matter to map out and depict accurately these abnormal conditions. We must, therefore, limit ourselves preliminarily to a simple statement of the clinical facts thus far known, and present, as well as we can, the leading points of view for the interpretation and treatment of these groups of symptoms.

1. ANACIDITY OF THE STOMACH (ACHLORHYDRIA).—ACHYLIA GASTRICA

Fenwick was the first to observe that there are cases of chronic and complete absence of any demonstrable secretion of gastric juice, independently of chronic gastritis or gastric carcinoma, but associated with an almost complete atrophy of the glandular apparatus of the gastric mucous membrane. The clinical picture in these cases displayed scarcely any marked gastric symptoms. Anorexia and moderate oppression after eating were almost the only indications of dyspepsia; but there were observed gradually progressive anæmia and emaciation, so that finally the general weakness, independent of any other local symptoms, resulted in death. Since then we have learned, particularly from the investigations of Martius, that the grave symptoms of these cases cannot possibly be caused by an atrophy and cessation of function of the gastric glands alone, but occur only when there is also present an atrophy of the intestinal mucous membrane.

It has been shown, and we have ourselves repeatedly found, that it is pos-

sible to have an achylia gastrica—that is, persistent absence of hydrochloric acid (and pepsin, *vide infra*), without any noticeable disturbance of nutrition, and with only slight and fugitive dyspeptic symptoms, or even with no gastric symptoms whatever. In all these cases it is wholly probable that the digestion and absorption by the intestines are perfectly normal, and completely replace the digestive function of the stomach. It is only requisite that the intestines should preserve their normal function, and that the motor power of the stomach should be unimpaired. In such cases the stomach serves to a certain degree merely as a reservoir for the ingested food, which it duly passes on to the intestines; in them are present in abundance all the reagents, bile, pancreatic juice, and intestinal juice, requisite for the further absorption of the ingesta. There is only one defect in these persons with achylia gastrica: there is no disinfection of the gastric contents by means of the hydrochloric acid of the gastric juice. Ordinarily this lack is unimportant, but if any hurtful material reaches the stomach it is more likely to lead to disturbance than under normal conditions. This explains why persons with achylia gastrica have a "sensitive" stomach and are prone to suffer from temporary dyspepsia. They very often also have a striking tendency to diarrhea. But if such individuals live cautiously and are not exposed to any especial injurious influences, they feel perfectly well and may be extremely well nourished.

Is there an atrophy of the gastric mucous membrane as a foundation for these benign cases of achylia gastrica? It is possible, and in many cases even probable, that there is; but it is not certain. This is certain: that sometimes a long persistent absence of hydrochloric acid has been observed clinically, and later an autopsy has shown no anatomical explanation of the condition. Thus, for example, a short time ago the author had a patient under observation in his wards for a considerable time with diabetes mellitus, in whose gastric juice no trace of hydrochloric acid could be demonstrated, although repeated examinations were made. The patient died, comatose, and the stomach seemed to be perfectly normal, even upon microscopic examination. Here there may perhaps have been some toxic inhibition of the secretion. At any rate, observations like this prove that when there is no gastric juice secreted, we cannot assert that any marked anatomical change will be found in the gastric mucous membrane.

From what has been said it follows that, according to the present state of our knowledge, we must distinguish several forms of achylia gastrica (or of long persistent gastric anacidity). We should mention incidentally that in a strict sense the name *achylia* should be employed only for that condition in which there is a constant absence not only of hydrochloric acid, but also of pepsin, indicating a complete drying up of the secretion of gastric juice. Those cases in which free hydrochloric acid is never demonstrable but pepsin is found should, in strictness, be termed anacidity, or, better, *aclorhydria*. In practice it is true that we cannot always make these fine distinctions, and that even such cases as present not an absolute absence but a slight or occasional trace of hydrochloric acid are classed in this latter category. We distinguish the three following forms of achylia:

1. Achylia or achlorhydria may appear as a symptom in certain other diseases of the stomach, particularly, as we have already seen, in severe acute and chronic gastritis (when there is genuine catarrh of the stomach), and also in cancer of the stomach. These cases are to be termed "symptomatic achylia."

2. Achylia or achlorhydria may appear as a necessary result of marked atrophy ("anadenia") of the gastric mucous membrane. Here the further point is to determine the causes of this distinctly characterized anatomical change in the gastric mucous membrane. Sometimes it seems to be the result of a previous catarrhal inflammation (atrophic catarrh, analogous to similar conditions of other mucous membranes—*vide supra,* page 492). How far this assumption agrees with the facts must be determined by further careful investigation. Apart from this secondary or inflammatory atrophy there is also a simple primary atrophy of the mucous membrane. The cause of this condition is most likely to be found in congenital peculiarities, of which we as yet possess no intimate knowledge. We might see a certain analogy in progressive muscular atrophy. In these cases of atrophy of the gastric mucous membrane there is no demonstrable secretion of hydrochloric acid, nor of pepsin. The gastric contents obtained in the ordinary way are absolutely devoid of peptic activity, but at the same time the muscular efficiency of the stomach is in many cases perfectly normal, and this explains why, although there is no secretion of gastric juice, there are no marked symptoms of gastric discomfort. In many cases, however, the atrophy of the gastric mucous membrane is coincident with an atrophy, or at least with marked changes of the intestinal mucous membrane. Then it is inevitable that severe disturbances of nutrition should gradually develop, for the food cannot be sufficiently digested and absorbed. Such patients grow progressively thin and feeble. Often they suffer from marked intestinal symptoms, having persistent diarrhea, alternating with constipation. By suitable treatment and nursing such patients may improve, or at least their symptoms may be checked. In a few rare cases death finally occurs. The clinical picture of pernicious anæmia (*q. v.*) has been repeatedly presented in association with this sort of atrophy of the stomach and intestines. In our opinion, however, the nature of these cases is still unsettled, since the atrophy of the intestinal mucous membrane of itself might well lead to extreme inanition, but not to a specific anæmia. For this there must be present some special circumstances with which we are as yet unacquainted.

3. The third form is simple functional achylia. Here belong the not infrequent cases in which the symptoms are not usually particularly severe, consisting of anorexia, gastric oppression, nausea, and rarely vomiting. The complaint may have been of recent origin. On careful examination of the gastric contents, we find complete absence of hydrochloric acid, while pepsin may be present, or, again, in other cases entirely absent. The motility of the stomach is normal, as a rule, but it may be somewhat impaired, probably because of a coincident weakness of the muscular coat (see the following chapter). With suitable treatment the symptoms diminish or vanish, but there is no change with regard to the absence, or approach to absence, of hydrochloric acid and pepsin. It cannot be determined how long this condition has existed. Sometimes such patients say, as we have already mentioned, that they have been rather subject to mild dyspepsia or to temporary attacks of diarrhea. Often, however, their previous health has been good. Whether we should invariably assume that there is a marked atrophy of the gastric mucous membrane in all such cases is very questionable. Probably we have rather to do with an impairment of the secretory capacity of the individual

stomach from causes not at present accurately determinable. Apparently there are not a few persons with achylia of this sort. Inasmuch as the condition runs along without any special symptoms, it is not diagnosticated by the physician, and the patient is not subjected to an accurate examination until digestive disturbance arises because of some special cause (*vide supra*), and then the anomaly is detected. If such patients do suffer from persistent dyspepsia there will usually be found some special reason for it, such as motor incapacity, possibly dislocation of the stomach, or genuine "nervous" dyspepsia, meaning by that term dyspepsia of psychical origin. It would be an interesting question to investigate whether individuals with persistent achylia do not perhaps have a predisposition to the development of carcinoma. I would like also to remark here that I have seen several accident cases with complaint of persistent and severe dyspeptic symptoms (pain, anorexia, etc.). These arose after a severe blow in the gastric region, and the examination of the gastric contents always showed an absence of free hydrochloric acid.

Diagnosis.—The diagnosis of achylia, whether essential or secondary, can always be easily made by means of a careful examination of the gastric contents (see the first chapter of this section). Of course, the diagnosis is not established until repeated and painstaking examinations have been made at various times and under various circumstances, and have invariably given the same negative result. If achylia is demonstrated, the next question is whether it is merely symptomatic or essential. The presence of chronic gastritis is excluded by the absence of all ætiological factors and of excessive mucous secretion, and by the general course of the disease. The suspicion of cancer will often likewise be rendered untenable by the general course of the disease and the absence of a tumor. In cases of atrophy of the gastric and intestinal mucous membrane, with consequent emaciation there may be very great difficulty in excluding cancer. On the whole, however, the course of gastro-intestinal atrophy is much slower than that of carcinoma. Accompanying signs of pyloric stenosis invariably indicate the presence of carcinoma; and if we are enabled by the absence of any severe disturbance of nutrition to exclude any marked atrophy of the gastric and intestinal mucous membrane, we reach at last a diagnosis of simple functional achylia by exclusion. Whether in such cases there is a demonstrable anatomical change in the gastric mucous membrane, the present state of our knowledge does not enable us to determine.

Treatment.—We do not need to enter upon a special discussion of the treatment of symptomatic achylia. The severe cases which occasion extreme disturbance of nutrition need the most careful dietetic management. They should have such food as milk, soups, eggs, and meat peptones. Practical experience will usually be a better guide than any theoretical considerations. Internally we may try hydrochloric acid and preparations of pepsin and pancreatin; also all sorts of stomachics, including nux vomica and condurango. The stomach may be rinsed out with a 0.2-per-cent solution of hydrochloric acid, and galvanic electricity may be employed. It is self-evident that when there is actual atrophy of the mucous membrane, the results of treatment must be very limited. The milder cases of achylia do not need treatment except when special dyspeptic symptoms appear, or when there is intestinal disturb-

ance, such as diarrhea; then the treatment is the same as in ordinary disturbances of the stomach or bowels, including a suitable diet, hydrochloric acid, perhaps rinsing out the stomach a few times, and, in case of diarrhea, opium. If the symptoms subside, and we now find there is a persistent achylia, of course we must earnestly recommend such individuals to exercise a certain caution with regard to diet. They should not take too great a quantity of food at one time, and in general should choose a largely vegetable diet —i. e., one containing an abundance of starches. There is this exception, that if the achylia is associated with a dread of eating and a tendency to hypochondriasis, we must encourage the patient to take a rather abundant amount of nourishment and employ hydrotherapy and other general tonics.

2. HYPERSECRETION AND HYPERACIDITY OF THE GASTRIC JUICE (ACID DYSPEPSIA)

We have seen in the first chapter of this section that it may be regarded as a rule that, when fasting, the stomach is almost completely empty, and that an hour after a test breakfast (*vide supra,* page 513) the acidity of the gastric contents is about 55 to 65. The investigations of late years have shown, however, that this rule has numerous individual exceptions not necessarily pathological. There are many persons who secrete scarcely any hydrochloric acid (*vide supra*), and also there are apparently still more who always have hydrochloric acid in the stomach even when fasting, and whose hydrochloric-acid index after a test breakfast is as high as 80 to 100, or even higher. Many such individuals have no dyspeptic disturbances whatever, so that the unusual abundance of hydrochloric acid can be regarded only as an individual peculiarity of secretion, and not due to disease. In other such cases, however, there are decided gastric symptoms, and these symptoms are such as to indicate very strongly their direct dependence upon the increased production of acid. This state is termed acid dyspepsia, by which, as already stated (page 525), we mean the abnormal excess of hydrochloric acid at the time of digestion— that is, hyperacidity as distinguished from hypersecretion, which latter term is applied to the continuous secretion of gastric juice even when digestion is not taking place, the stomach having received no food, or having discharged its contents into the pylorus. Hypersecretion is probably almost always associated with hyperacidity, but not *vice versa.* However, we cannot maintain a strict clinical distinction between the two in practice.

Essential, acid dyspepsia should be sharply distinguished from symptomatic hyperacidity and hypersecretion, such as occur perhaps in certain forms of gastritis (page 523), and almost invariably in ulcer of the stomach. This distinction is very important, even from a practical point of view. It must be confessed that it may be scarcely possible to make a differential diagnosis, as we shall soon see, between ulcer and hypersecretion; but we can, at any rate, hold fast to the important theoretical distinction between the two conditions. The term "essential" hypersecretion ought to be applied only to cases in which there is an increase in the secretion of hydrochloric acid, without any ulcer formation. If we regard hypersecretion as a possible cause of ulcer (*vide supra,* page 530), then ulcer can be termed merely a complication of hypersecretion.

As to the causes of simple hyperacidity and hypersecretion we have as yet, unfortunately, scarcely any knowledge. Very few histological examinations of the mucous membrane in characteristic cases have thus far been made. It is, therefore, at present customary to assume that hypersecretion is a "neurosis," and this view is apparently confirmed by the frequent combination of hypersecretion with other nervous conditions. We must later discuss this point more fully. On the other hand, however, it is not impossible that functional disturbances of cellular activity may lead to increase in the secretion. We are ourselves inclined to class many cases of hypersecretion along with the well-known cases of increase in the secretion of sweat (hyperidrosis of the hands and feet), of saliva, and of similar products. For such it is not at all essential that there should be abnormal nervous conditions.

Having such imperfect insight into the true nature of these processes, we must provisionally take a purely clinical standpoint, and describe the phenomena seen in practice. It should be added that some of the conditions are by no means infrequent, but it is often difficult to interpret accurately what we observe. One important question is how far the symptoms which are present are actually referable to the hypersecretion as such, and how far they are due to the other anomalies which often accompany the hypersecretion, such as general nervousness, gastroptosis, and chlorosis. In order to get a better comprehension of the subject we shall distinguish in what follows several forms of acid dyspepsia, but we wish to repeat that a perfect differentiation is impossible, particularly between hyperacidity and hypersecretion.

1. Dyspepsia with Hyperacidity (Hyperchlorhydria).—The conditions which belong in this category are most often found in young females. They are very often conjoined with symptoms of chlorosis, general nervousness, etc. Certain errors in diet may also excite hyperchlorhydria, such as hot or highly spiced food, and particularly too strong coffee; and dyspeptic conditions with hyperchlorhydria are frequently observed in excessive smokers.

The symptoms are tolerably characteristic. They occur especially after eating, and sometimes only after certain kinds of food, and consist first of a sense of oppression in the stomach, often increasing to genuine cardialgia. Usually the pain does not appear until two or three hours after eating—i. e., at a time when the stomach is already empty, although the excessive secretion of acid is still going on. Many patients have noticed that if they then take some more food or drink their pain will abate. This is easy to understand. Often there will be acid eructations at the time of the excessive formation of acid, and a distinct feeling of heartburn. The acid gastric contents may be vomited, but this is not especially frequent. When the stomach is completely empty, the patient feels perfectly well again. Inasmuch as the appetite is usually unimpaired, nutrition remains fairly good. The general course of the disease is chronic, with marked variations. At many times the symptoms are aggravated, particularly under the influence of psychical disturbances, such as anger or excitement, or because of an unsuitable mode of life. At other times the symptoms may entirely vanish.

The diagnosis of hyperchlorhydria may be suggested by the symptoms just described, but it cannot be established except by means of the stomach tube. External examination of the stomach shows nothing special except that there may be a moderate diffuse tenderness on pressure. It is not surprising that

often there is gastroptosis. If we examine the stomach when fasting, it is empty or almost empty, provided the case is one of pure hyperchlorhydria. The acid index after a test breakfast is very high, 70 to 100 or over. The digestive power of the gastric juice is greater than normal. It is therefore natural to find, as we do find, that the stomach is entirely empty, as a rule, as soon as three or four hours after a test meal, which indicates also a good degree of motor power. At that time hydrochloric acid is still present. On the other hand, digestion of starch in the stomach is impeded by the abundance of hydrochloric acid.

If we find things as above depicted, the diagnosis of hyperchlorhydria as such is established. But a difficult question remains to be answered—whether the case is one of simple functional increase of the secretion, or whether the hyperacidity is symptomatic of an ulcer. If there are no characteristic symptoms of ulcer, such as gastric hemorrhage and localized tenderness, it is often impossible to answer this question with more than a certain degree of probability. It is characteristic of simple hyperchlorhydria that the pain is often improved upon taking food, while in ulcer the pain is aggravated. If there are well-marked symptoms present of general nervousness, this is usually decidedly in favor of "nervous hyperacidity." Finally, the results of treatment may aid us in diagnosis (*vide infra*).

The mode of treatment will be considered with that of hypersecretion.

2. Dyspepsia with Hypersecretion of the Gastric Juice ("Continuous Flow of Gastric Juice").—Here belong those cases of dyspepsia in which the examination of the stomach shows the presence, even when fasting, of considerable amounts of fluid containing an excess of hydrochloric acid, and in which this hypersecretion cannot be regarded as the result of an ulcer.

The disease is much more exceptional than simple hyperchlorhydria. It appears in men somewhat oftener than in women. No special ætiological factors can be named, except long-continued mental strain and excitement; often no cause can be made out.

Symptoms.—The symptoms, as in hyperchlorhydria, consist of cardialgia, sour eructations, heartburn, and not infrequently vomiting. Pain often appears when no food has been taken. When vomiting occurs there may be little or no food in the vomitus, but merely a cloudy, often bile-stained fluid, strongly acid, with a very sharp taste and so irritating that it causes burning pain in the pharynx. The appetite is generally good, and the patients have usually found out by experience that if they drink some tea so as to dilute the gastric juice, or take some sort of food, they get relief. Often there is abnormal thirst; the bowels are usually constipated; the urine faintly acid and turbid with deposits of phosphates; the pulse is often slow; in many cases there is a distinct general neurasthenic condition, associated with the dyspepsia. Here, again, the only method of arriving at a certain diagnosis is by examining the gastric contents. The decisive point is that in the morning before breakfast we should invariably find a considerable amount of fluid, containing practically no portions of food, but a large percentage of hydrochloric acid. At the same time it should be particularly pointed out that it is not abnormal, now and then, to find a teaspoonful or two of fluid in the stomach, containing hydrochloric acid, while fasting. It is not proper to speak of the case as one of hypersecretion unless we can obtain from the fast-

ing stomach, without special effort, some two or three ounces (50 to 100 c.c.) of strongly acid fluid. Along with the hypersecretion there is usually hyperchlorhydria, so that we find a high acid index an hour after the test breakfast. If we give a test meal, and examine three hours later, we shall find that the meat has vanished, while the digestion of starches has been decidedly delayed by the hyperacidity. Some seven hours after the test meal the stomach is empty of food, but probably contains an abundance of acid secretion.

If we inflate the stomach, we may find the organ dislocated downward (gastroptosis). This, however, is merely a chance complication. There is never great dilatation. If we find the stomach much enlarged, our suspicions are at once directed to pyloric stenosis, and this condition is usually easily diagnosticated, one important indication being delay in the emptying of the stomach. Whenever the stomach in a fasting condition, or seven or eight hours after a test meal, is found to contain not only an abundance of hydrochloric acid, but in addition considerable amounts of partially digested food, the condition is not one of simple hypersecretion, but of hypersecretion combined with stenosis of the pylorus due to ulcer. This puts an entirely different aspect on the case. The assumption that, in the presence of hypersecretion, a marked retardation in the emptying of the stomach can be produced by a purely functional cramp of the pylorus, I consider entirely unproved. In all my cases of hypersecretion and pronounced disturbance of the motor activity of the stomach, an ulcer was eventually demonstrated with certainty, even if in the beginning it seemed reasonable to assume the presence of a purely functional hypersecretion.

It is a much more difficult matter to distinguish hypersecretion from a gastric ulcer without stenosis. Indeed, sometimes an absolute decision is almost impossible. In such instances we may be enabled to form an opinion by considering the general course of the disease, the associated nervous symptoms, and all the special symptoms (*vide supra*).

In general, the course of acid dyspepsia with hypersecretion is chronic, but variable. The prognosis is not unfavorable, and correct management, based, as it must be, upon an accurate examination, may bring about very satisfactory results.

Treatment.—In the treatment of acid dyspepsia, including hypersecretion as well as simple hyperchlorhydria, we meet with one difficulty in the fact that we can seldom absolutely exclude ulcer of the stomach. The physician must, therefore, determine whether he shall begin with a course of treatment for ulcer or not. We believe that in all cases in which the symptoms have existed for a considerable time and are severe, and in which there has been no systematic treatment as yet, it is wise to treat the patient as if there were an ulcer. If the case is one of simple acid dyspepsia this can do no harm, and if it is one of ulcer it is the only method that can do good. In either case the quiet, the unirritating diet, and the administration of alkalies are certainly appropriate. Moreover, cases are not infrequent in which the patients are anxious and hypochondriacal, and upon them a methodically conducted "cure" for ulcer often has a favorable subjective influence. Still, this just-mentioned combination of acid dyspepsia with decided nervous and hypochondriacal disturbances may, on the other hand, render the effect of a strict ulcer treatment quite unfavorable. This applies to patients who have

long been tortured by a dread of gastric ulcer, have consequently gone through the appropriate treatment many times already, and have grown thin and feeble as the result of an excessively cautious dietary. For such unfortunates there may be no use whatever in repeating a strict course of treatment suitable to ulcer. Such a procedure confirms the patient in his apprehensions, and merely makes him more wretched.

We see, then, how carefully we should consider the individual in the treatment of acid dyspepsia, and, above all, how much weight we should assign to the general condition as well as to the dyspeptic symptoms. It is often advisable to begin with a course of treatment for ulcer in the ordinary manner, but upon the prompt cessation of symptoms to make a more rapid transition than usual to nourishing food and other therapeutic methods. The more convinced the physician becomes that he has to do with simple "nervous" hypersecretion and not with an ulcer, the less need has he to pursue a strict dietetic treatment. In such cases it is best to prescribe for the patient a diet list, containing an abundance of albuminoids, including meat, fish, eggs, and milk; and with this, rye bread and butter, and easily digested vegetables. There should be as great a limitation as possible of alcoholic beverages, spices, strong coffee, and sour articles, as well as of puddings and starchy vegetables. Yet in such cases, also, one must be guided by the individual experiences of the patient as well as by merely theoretical considerations. As to drugs, of course alkalies come first. We should be guided by the circumstances of the particular case in prescribing, several times a day, particularly after meals, a knife-point full of bicarbonate of soda, or some similar powder, such as bicarbonate of soda and calcined magnesia, equal parts; or bicarbonate of soda, 30 parts; subnitrate of bismuth, 5 parts; and similar drugs. If there is hypersecretion we like to give the patient in the morning, before breakfast, a half liter (pint) of warm Carlsbad water, or the artificial Carlsbad salt, dissolved in water. In severe cases of hypersecretion regular washing out of the stomach is advantageous. The best time to do this is in the early morning. We may employ a one-per-cent solution of bicarbonate of soda.

Atropin and the narcotics, such as morphin and codein, have been recommended for the purpose of diminishing the secretion of the gastric glands. We have used both belladonna (for example, extr. belladonnæ, gr. v [gm. 0.3]; aq. amygd. amar., 1 ounce [30.0 c.c.]; 15 to 20 drops t.i.d. p.c.) and atropin with success, but we would warn the physician against the unnecessary employment of narcotics.

The simultaneous constitutional treatment is very important, particularly in all cases associated with neurasthenic symptoms (*vide infra,* the chapter on Nervous Dyspepsia). From this point of view there is much benefit in drinking the waters at Carlsbad, Tarasp, and similar watering places, in connection with the hope of constitutional improvement from the outdoor life, the baths, cold sponging and rubbing, and similar agencies. In treatment at home these last factors must always be duly considered.

3. **Periodical (Intermittent) Hypersecretion (Gastroxynsis).**—In the condition here to be described, persons who at other times feel perfectly well, without any digestive disturbance, are rather suddenly attacked by violent gastric pain, sour eructations, and vomiting. The intervals between attacks may be

longer or shorter, and there may be either some exciting cause or no apparent reason for the disturbance. The vomitus consists of large amounts of watery fluid containing an excess of hydrochloric acid. During these attacks the general condition of the patient is wretched; he looks pale, his pulse is small and frequent. Often there is extremely violent headache at the same time. An attack of this sort lasts a few hours, or even two or three days. Often it ends with considerable suddenness, to be followed by a period of good health which may last a week or even months, or longer.

The causes of this strange disease are entirely unknown, but it certainly seems as if there were conditions of irritation, originating in the nervous system. It seems to us indubitable that there is a close relation between gastroxynsis and migraine (see the chapter on Migraine). It is a very important fact that almost precisely the same group of symptoms occurs in tabes dorsalis (*q. v.*), under the name of gastric crisis. In every case, therefore, the physician should examine the tendon reflexes and the pupils. In pyloric ulcer with stenosis attacks may also occur that at first simulate a nervous gastroxynsis. I have no doubt whatever about the occurrence of a purely nervous form of this malady, although in its well-marked form the condition is quite rare.

If possible we should examine the gastric secretion in the time between attacks, as well as during the attack. It appears that many cases of periodical gastroxynsis are merely acute exacerbations of a constant hypersecretion. In such cases there are likely to be mild dyspeptic symptoms in the intervals, and a careful treatment of these by lavage, alkalies, and Carlsbad water has an excellent effect upon the paroxysms. If there are coincident neurasthenic symptoms, of course the greatest stress must be laid upon constitutional treatment. The treatment of the paroxysm itself is seldom very successful. The best remedy is bromide of sodium in large doses. We may also try chloral, chloroform, belladonna, codein, antipyrin, and coryfin (6 to 10 drops). Externally, warm compresses or hot poultices may be applied over the stomach. If the pain is very violent a subcutaneous injection of morphin may be almost indispensable, although we should avoid it if possible.

CHAPTER VIII

ABNORMALITIES IN THE SIZE AND POSITION OF THE STOMACH—MOTOR DISTURBANCES OF THE STOMACH

1. ABNORMALITIES IN THE SIZE OF THE STOMACH—ATONY OF THE STOMACH

Like any other organ, the stomach may present considerable differences in its size without being pathological. These differences partly depend upon congenital conditions, and partly are associated with the mode of life. It has long been known that certain classes of people who eat very bulky articles of diet (e. g., a great proportion of potatoes), and other large eaters, including many diabetics, have noticeably large stomachs; but a change in size of this sort cannot be regarded as pathological, provided the function of the stomach is not impaired. The excessive distention of the organ, or the excessive demands

upon the functional activity of the muscular coat of the stomach, may finally render it unequal to its task. Then we reach a pathological disturbance, a sort of overstrain of the stomach, a muscular insufficiency analogous to the myopathic diseases of the heart, which have been so much studied. Obviously one important factor here would be the original quality of the muscular fiber: if the muscles of the stomach are weak from the start, they will become insufficient all the sooner.

We must state that in our experience well-marked cases of primary idiopathic dilatation of the stomach, as an expression of pure muscular insufficiency, seem to be far from frequent, although slight degrees of muscular weakness occur quite often.

From what has been said, it is evident that the physician should lay little stress upon the mere estimation of the size of the stomach. And, indeed, its size varies greatly according to the amount ingested. We can form an opinion of the size of the organ by percussion of the empty stomach, or by percussing it alternately empty and filled with water by means of the stomach tube, or, what is much simpler and more certain, by distending it with air, as above mentioned (page 515). In general, we can say there is dilatation of the stomach if the lower border of the fundus extends below the level of the navel while the lesser curvature is in a normal position; but, as we have said, the important question is not the size, but the motor ability of the organ, and it is far more important to determine the functional activity by means of a test meal, in the manner already explained (page 514), than to find out the mere size of the stomach. If the organ empties itself in a normal way, any dilatation that there may be has no significance. If there is some tendency to stagnation of the gastric contents because of muscular insufficiency, we may have mild dyspeptic symptoms, such as gastric oppression, eructations, or even occasional vomiting.

If, however, we find that the stomach is dilated and that the motor efficiency is decidedly impaired, with actual stagnation and accumulation of ingesta in the stomach, we ourselves think it is always justifiable to assume that there is a pyloric stenosis. In such case the disease with which we have to deal is not a dilatation of the stomach, but an ulcer or cancer of the pylorus, which leads to stenosis and its results, as we have already described at length (pages 537 and 547). It has often been maintained that in cases of primary muscular dilatation there may occur mechanical kinks in the pylorus and sacculations in the stomach, so as to cause a considerable hindrance to the emptying of the viscus. This may be possible, but we have never seen such a case, nor read a conclusive report of one.

In this place a symptom may be mentioned which is often regarded as a sign of dilatation. We refer to splashing in the stomach when palpation is practiced by a sort of pushing or thrusting motion. Marked splashing is indeed very often audible in cases of actual dilatation due to stenosis of the pylorus, but it is very frequently heard in persons who have dyspepsia, and even in persons who are perfectly well. Children sometimes play at producing splashing sounds in their own stomachs, and by practice acquire great technical dexterity! And in precisely the same way the sound is produced by nervous and hypochondriacal patients, to whom the splashing is a source of anxiety. Splashing occurs when air and fluid are both present in the stomach,

provided the abdominal walls as well as the gastric walls are not too tense. In this sense it is, therefore, correct to associate so-called atony of the stomach with the splashing, because it may often be due to a certain laxity of the muscular coat of the stomach, but we should never lay any special stress upon the sound, unless upon examination we find at the same time a distinct disturbance of the motor efficiency of the stomach.

It follows, therefore, that mere dilatation of the stomach is not an independent disease. In practice we may unhesitatingly refer every case of well-marked dilatation with decided stagnation of the gastric contents to stenosis of the pylorus, and of course this latter is to be regarded as the true disease. At the same time, however, there are slight degrees of gastric dilatation, or perhaps a better expression would be idiopathic motor insufficiency of the stomach (muscular atony of the stomach), which occur when there is no reason for believing that there is any anatomical change at the pylorus. We may assume that such motor insufficiency exists when there is a slight but distinct tendency for food to accumulate in the stomach.

In treating this simple muscular atony, we should first regulate the diet. The separate meals must not be too large, but they should be nutritious, and perhaps even somewhat stimulating. Rye bread, small portions of vegetables, and nourishing meat dishes are by no means interdicted. Large amounts of liquid are to be avoided, as they needlessly dilate the stomach. In simple atony, lavage is seldom necessary. At the most, we might at the beginning of treatment completely empty the stomach a few times. Other suitable measures are cold sponging and rubbing of the epigastrium, the application of electricity (particularly the galvanic current) to the stomach, and cautious massage of that region. Among internal remedies, nux vomica deserves to be employed.

Whether there is such a thing as abnormal smallness of the stomach, giving rise to symptoms, is not yet known. Perhaps there may be such a condition in cases in which feeble and ill-nourished individuals, usually of the female sex, can take but a few morsels before they have a feeling of fullness and satiety, and so never become well nourished despite every effort. We may find the stomach very small as a secondary result in many forms of inanition, particularly in association with stenosis of the œsophagus and of the cardia.

2. ABNORMAL POSITION OF THE STOMACH—GASTROPTOSIS

Kussmaul was the first to call attention to the great frequency of the downward dislocation of the stomach. Glénard has written much about it. Either the entire organ may sink down, or, what is more common, the right half alone, so that the organ takes a vertical position. The cause of this anomaly may lie partly in congenital conditions, but in the main it is certainly the purely mechanical result of the pressure of clothing. Gastroptosis is an extremely common phenomenon in grown-up girls and women who wear tightly laced corsets, and fasten the bands of their clothing snugly about them. It also occurs in men, but far less often. Usually, with the gastroptosis is associated a change in the position of other organs, occurring in the same way. In particular, the right kidney becomes abnormally movable, and consequently readily palpable (see the chapter on Movable Kidney), and the transverse colon sinks down

either in its right half or in its middle portion. Glénard has termed this condition enteroptosis, and ascribed to it an important rôle in the ætiology of nervous dyspepsia and neurasthenia. Well-marked cases of enteroptosis are most frequent in women who have lax abdominal walls because of repeated confinements.

The experienced eye may often recognize, or at least surmise, gastroptosis upon external inspection of the abdomen. One is struck by the collapsed appearance of the epigastrium, where one often feels marked pulsation of the abdominal aorta. Below the navel, on the other hand, is seen a protuberance, the shape of which may quite distinctly suggest the contours of the stomach. At this spot, below the navel, distinct splashing may not infrequently be obtained by proper palpation. An absolute diagnosis of gastroptosis, however, requires the artificial inflation of the stomach (*vide supra,* page 515) or an X-ray examination of the stomach filled with bismuth paste. Of course, the most important point is the demonstration of a downward displacement of the lesser curvature. This is found halfway between the navel and the epigastric angle, or often even at the level of the navel. The downward displacement of the greater curvature alone does not establish gastroptosis, because it may be due to dilatation of the stomach.

Inflation of the stomach is such a simple and reliable method of examination that there scarcely seems any real necessity for other methods. For this reason Einhorn's recommendation of electric transillumination of the stomach, by means of an incandescent light introduced into the organ ("gastrodiaphanoscopy"), is scarcely likely to become generally adopted by physicians. It gives some indications of the position of the stomach, but the conclusions drawn from its use are by no means infallible. The displacement of the kidney, which is often associated with gastroptosis, can be easily recognized by means of palpation. The position of the colon (coloptosis) can be demonstrated by distending the large intestine with air or water.

The question of the clinical significance of gastroptosis is not very easy to answer. In the first place, it should be remarked that, if we pay attention to the subject, we shall find that well-developed gastroptosis is very frequent in persons who have not the slightest symptoms. On the other hand, there are many patients, especially women, who complain of such annoyances as pressure and a sense of fullness, attacks of gastric pain, eructations, and occasional vomiting, and who are found to have a distinct gastroptosis. In such a case we should invariably proceed to test the secretory and motor functions of the stomach. Very often these are perfectly normal, and such a condition scarcely justifies us in assuming offhand that there is a causative relation between the gastroptosis and the patient's symptoms. Gastroptosis is so common that the relation is more likely one of simple coincidence. If we make a searching examination into the patient's condition we shall usually discover that the gastric symptoms are associated with many other symptoms of a neurasthenic kind, and hence are very likely, for the most part, of purely psychical origin (see the following chapter).

Still, it cannot be absolutely denied that certain disagreeable sensations in the abdomen and along the œsophagus (perhaps due to the traction exerted on it) may be directly dependent upon gastroptosis. Furthermore, gastroptosis in some few cases seems to occasion a mechanical hindrance to the discharge

of the stomach contents, especially because of the vertical position of the organ, so that we have a sense of oppression in the stomach, eructations, and other symptoms; but this motor insufficiency should be proved, not assumed. As a rule, even well-marked gastroptosis is not associated with any disturbance of the motor function of the stomach. There is more likelihood that the habitual constipation, which often accompanies the dyspeptic symptoms of gastroptosis, is at least in part the result of an associated coloptosis.

Treatment.—For the treatment of the dyspeptic symptoms which may be present in a case of gastroptosis, we have a most desirable basis in the gastroptosis itself, although the author at least is convinced that its main efficacy is subjective. If gastroptosis has been made out, we must absolutely forbid the wearing of tightly laced corsets or too snugly fitting gowns. It is often useful to apply a wide abdominal band with leg straps above the symphysis. This in many cases fully replaces the more complicated "abdominal corsets" and similar apparatus. With regard to diet, we forbid the taking of large amounts of liquid, and prescribe nourishing food that is not bulky. Particularly when patients are emaciated and apprehensive, we often do the greatest good by ordering a highly nutritious diet. If the emaciation is marked, and there is general weakness, a complete rest cure for several weeks (lying in bed or on a sofa at the open window or outdoors, without any constricting clothes), with careful forced feeding at the same time, is often attended by remarkably good results. The region of the stomach should be rubbed briskly once or twice a day with cold water or brandy. The employment of faradic and galvanic electricity or massage is particularly indicated when constipation is present. We often prescribe small doses of nux vomica internally (e. g., tr. nucis vom. and aqua amygdal. amar., equal parts. Ten drops of this to be given thrice daily).

Of course we must pay attention, as we have said, to the general neurasthenic condition, which is usually also present. In this regard great importance attaches to a suitable mental treatment, in order to free the patient from her exaggerated anxiety and apprehension.

CHAPTER IX

NERVOUS DYSPEPSIA

(Gastric Neurasthenia)

We have repeatedly indicated in the preceding chapter that many disturbances of the gastric functions may be due to changes in the innervation of the stomach, or to actual disease of its nerves; thus some have been inclined to refer the disturbances of the secretion of the gastric juice, such as hyperacidity and hypersecretion, to abnormal conditions of the secretory nerves. Many cases of "cardialgia" have been regarded as pure "neuralgia" of the sensory nerves of the stomach; and also in the domain of the muscular activity of the stomach, authors have distinguished conditions of nervous weakness (nervous atony of the stomach) and of nervous irritability of the muscular coat (so-

called "peristaltic unrest" of the stomach; constant rumbling in the stomach and similar symptoms). There is no doubt that in cases of severe organic disease of the nervous system there may be well-marked gastric symptoms as direct consequences of nervous irritation—for example, we have nervous vomiting in cerebral tumor and meningitis, and gastric crises in tabes. It is, therefore, not impossible that there may also be primary diseases of the sensory, motor, and secretory nerves of the stomach with corresponding symptoms, but this is by no means proved, and it is certain that such actually "nervous" gastric diseases are very rare, if they occur at all. At present, clinical experience does not justify us in putting in this category, with any degree of certainty, any disorder except the state above described (page 562), in which there is periodical vomiting with gastric pain and hypersecretion.

There are numerous cases which are at present habitually termed nervous dyspepsia, but in our opinion the overwhelming majority of these have an origin different from the one implied. Such patients assert that after every meal they have pressure and pain in the stomach, that after a few mouthfuls the stomach feels full and distended, and consequently they often have palpitation and a troublesome feeling of constraint in the chest. The patients frequently complain of eructations also, and occasionally of vomiting. These symptoms are all such as occur in the same way in actual diseases of the stomach, but in these patients there are usually certain other symptoms present, which indicate the nervous character of the disease. In the first place, it is noticeable that the above symptoms display striking variations in their severity. The same patient who to-day complains of great distress in his stomach after a few spoonfuls of soup, at another time, when he is in a happy and lively frame of mind, will eat a large dinner without being reminded that he has a stomach; but as soon as he gets angry or irritated, or, in brief, in a "sensitive" condition, the gastric symptoms appear at once in an exaggerated form. On all such occasions, therefore, we see most clearly the influence of the imagination and of general psychical irritation on the behavior of the stomach. Every one knows that a violent rage or any experience arousing anxiety or hope—any excitement, sad or joyful—may immediately destroy the appetite; and very great mental disturbance not infrequently also causes vomiting or similar symptoms. In the same way in sensitive individuals even the mildest psychical influences produce similar results; but in this regard there is nothing which has so unfavorable an influence as that state of psychical disturbance which is associated with great concern for one's own health—that is, a condition of hypochondriacal anxiety—anxiety lest something which has been eaten may do damage; constant dread that a grave disorder of the stomach may be developing. These mental disturbances are especially influential in maintaining and progressively aggravating the abnormal condition. This is the explanation of that peculiar psychical hyperæsthesia which feels the acutest "pain" in the stomach, when there is really nothing but a perfectly normal general sensation. And the same explanation applies to certain muscular contractions—half unconscious, half voluntary—which occasion eructations, vomiting, and similar phenomena. The patient has some symptoms which he feels subjectively, and some which seem to him to be actually objective, and they are in part the result of pure imagination (or "autosuggestion") and in part the physiologically necessary consequence of the great psychical disturbance.

What we wish to emphasize is our conviction that in the great majority of cases of nervous dyspepsia we are not dealing with any functional disturbance of the gastric nerves, but with abnormal "psychogenetic" irritations of the central nervous system, the results of which are apparent mainly in the domain of the gastric functions. Nervous dyspepsia is merely one example of that great group of nervous diseases which owe their origin mainly to hypochondriacal disturbances of mind, and which may appear in the most diverse organs. It is properly nothing but a symptom of constitutional "nervousness" or neurasthenia, and is therefore termed by many physicians, with absolute correctness, gastric neurasthenia. By careful clinical observation it is easy to demonstrate that almost all the ordinary gastric symptoms may, under favorable circumstances, be excited by purely psychical influences. It is evident that anorexia is often the result of simple mental disturbance, especially when of a hypochondriacal character; but excessive craving for food (bulimia) is certainly in most cases also of purely psychical origin, arising from special imperative conceptions. There is no doubt that the most varied abnormal sensations, ranging from simple oppression to a most acute pain in the stomach, may have a purely subjective origin. The best proof of this lies in the often apparently miraculous effect of remedies which have a purely suggestive and psychical influence (simple reassurance of the patient, hypnotism, and various external and internal remedies). It is very important to know that in many cases also eructations and vomiting have a merely psychical and central origin. They are due to the influence of certain ideas which make the eructations or vomiting appear unavoidable, and the action at first is due to unconscious volition, and finally becomes a sort of diseased habit. Many nervous and hypochondriacal patients who regard themselves as dyspeptics become true *virtuosi* in eructations and vomiting, the reflex process becoming easier and easier because of repetition, and being excited by unconscious volition. General psychical disturbance may also cause eructations and vomiting by direct stimulation. We know persons who are attacked with nausea and vomiting upon any great excitement. Even in children it is by no means rare to observe persistent vomiting after almost every meal, and that this is of a purely "nervous" character appears from the rapid beneficial effect of exclusively psychical treatment—e. g., if the child is strictly forbidden to vomit. We often see just the same condition in adolescent girls, frequently associated with other hysterical symptoms. In this connection it should be briefly mentioned that there may be a "nervous"—that is, hysterical—hematemesis. (For particulars in regard to this, the reader is referred to the chapter on Hysteria; and, in general, the chapters on Hysteria and Neurasthenia should be referred to as supplementing what has been said here.)

Very often other nervous symptoms are observed besides those referred to the stomach. There are signs of excessive mental irritability, symptoms referable to the head, such as headache, pressure in the head, vertigo; abnormal sensations in the extremities, of pain, coldness, numbness, and the like. There are almost invariably, also, certain attendant intestinal symptoms. The patient complains of bloating of the abdomen, and of irregular and sluggish bowels.

Nervous dyspepsia becomes a more serious matter when the patient is prevented from taking sufficient nourishment, by fear of committing errors in diet, and because of the loss of appetite which his mental uneasiness occasions.

There are sometimes complete nervous anorexia, a constant dread of eating or an actual aversion toward any food, and a considerable or excessive emaciation, usually associated with great bodily weakness. Such patients finally take to their beds and give the impression of severe illness.

Diagnosis.—In many cases the experienced physician can make an extremely probable diagnosis of nervous dyspepsia merely from the symptoms which the patient enumerates. The true condition is suggested by the general nervousness, by the prominence of anxiety and of hypochondriacal notions, occasionally the occurrence of marked attacks of fear with strong general psychical disturbances, the variability of the symptoms and their relation to states of consciousness (excitement, on the one hand, distraction and diversion on the other), and the other attendant symptoms of a nervous character, such as headache, vertigo, palpitation, and a sense of oppression. Still, it is easy to be deceived, because the symptoms seem to be so objective that the physician feels constrained to believe that there is some organic disease of the stomach, although none exists, and because, on the other hand, an actual disease of the stomach may be present in a very nervous individual and fail to be recognized, being wholly obscured by the general nervous phenomena. For this reason, even in cases which seem to be the most simple, we would do well to make a careful objective examination, and this is, of course, an absolute necessity in all cases which are obstinate and severe.

Often the objective examination shows clearly normal conditions in every respect: the external examination is normal, and the position, secretion, and motility of the stomach are normal. In such a case the diagnosis is established, and the mere result of examination will often have a most beneficial effect upon the patient. For this reason a painstaking examination is often the best remedy for many patients with nervous dyspepsia. It is more difficult to reach a conclusion when certain abnormalities are found, although there are no signs of indubitable organic disease—that is, there is no tumor nor evidence of stenosis of the pylorus nor genuine hemorrhage—but we do find, for example, distinct hyperacidity or hypersecretion; less often achlorhydria; very frequently gastroptosis; and often, it is said (although, according to the author's experience, this is rare), some slight disturbance of motility, so-called atony of the muscular coat of the stomach. How shall such cases be interpreted? Shall we always lay stress exclusively upon the objective anomalies which are discovered, and refer to them every one of the many symptoms the patient enumerates? In the author's opinion, this would often be erroneous. It has been especially emphasized in the preceding chapters that the conditions mentioned are frequent and are apt to cause no symptoms at all, so that if we find associated with well-marked nervous dyspepsia a gastroptosis, or a moderate hypersecretion, or achlorhydria, in many cases the association is a mere chance coincidence without significance. We think that such conditions should not be wholly disregarded, but yet their clinical importance should not be overestimated; and from a therapeutic point of view, also, they should be borne in mind, but never to the neglect of that general psychical treatment which is usually much more important. Such conditions also are very useful as a handle for mental therapeutics (as we have already pointed out), because of the favorable effect which treatment of them has upon the mind of the patient.

A most difficult question in diagnosis, in our opinion, is to distinguish between ulcer and nervous dyspepsia when there is distinct hypersecretion without evident signs of ulcer (see page 532). In such a case all the separate symptoms must be duly considered, and yet we may be obliged to await the further development of the disease and the results of treatment before arriving at a conclusion. Patients with nervous dyspepsia are apt to grow worse and worse under strict treatment for ulcer, while an opposite mode of treatment (*vide infra*) often has the most brilliant results.

Prognosis.—The prognosis depends mainly on the strength of the general neuropathic tendency in the case and on the surroundings in which the patient lives. If the patient is amenable to an appropriate suggestive treatment, brilliant therapeutic results can be readily obtained in "nervous dyspepsia," especially in those cases in which the patient has previously become extremely emaciated through a too rigid diet, and subsequently rapidly gains in strength and weight on a correct diet. If, however, the abnormal concepts and the excited general psychical condition are too firmly rooted to be readily disposed of, only slight and uncertain results are obtained. Likewise, a permanent improvement can rarely be expected in those cases in which the injurious psychical causes or other ætiological factors continue, while the removal of these causes will, on the contrary, frequently result in a complete cure even of apparently very grave conditions. A liability to relapses is, of course, almost always left behind, as most patients are constitutionally nervous.

Treatment.—If nervous dyspepsia has once been diagnosticated, the proper aim of our therapeutic efforts becomes perfectly definite. We must, in the first place, convince the patient that he has no incurable or even dangerous gastric disease, but that, on the contrary, his stomach is perfectly capable of performing its functions in a normal manner. Nothing could be more harmful to a sufferer from nervous dyspepsia than to have his physician manifest great anxiety about treatment, and prescribe a very strict diet. The patient must rather be gradually led to use an abundance of nutritious food. It is in this way alone that he regains confidence in himself, when he sees that the hearty food does him no harm, that he is gaining flesh, and that the bowels are becoming regular.

Internal remedies are best omitted altogether if the patient has already taken a good deal of medicine. If they are successful, it is usually because of their influence on the mind. If we must prescribe some medicine, the bitter tonics (the preparations of quinin, tincture of nux vomica, etc.) are most suitable. They serve particularly to stimulate the appetite. If painful sensations in the stomach are experienced after eating, we attempt to relieve them with warm applications or a Priessnitz bandage, twenty-per-cent alcohol applications, by administering some hydrochloric acid, pepsin, etc.; here, as before mentioned, suggestive influence is naturally of paramount importance. In this respect, the galvanic current (large anode to the region of the stomach, cathode to the back) is often particularly efficacious. The treatment of continuous ("habitual") nervous vomiting is very important, but frequently difficult. In cases of this sort also psychical treatment is of the greatest importance. Even if we gravely command the patient not to yield to the impulse to vomit, but to resist it as much as possible, such an injunction will often do

away with the symptom, and it is not rare to find in cases of nervous vomiting that it will cease as soon as nourishing diet is allowed, although previously it has occurred invariably after the most delicate kinds of food. It is often very advantageous in nervous vomiting to limit, as far as possible, the amount of liquids, such as soup and milk, and to prescribe a dry substantial diet. In many cases the vomiting will then cease very promptly.

Obstinate cases of nervous vomiting, it is true, are cured with difficulty at home, while they can frequently be rapidly relieved by the psychical influence of good sanatorium or hospital treatment. If, in addition to the purely nervous symptoms, certain objective symptoms (anæmia, gastroptosis, habitual constipation, etc.) are present, these will also have to be considered to a certain degree (*vide supra*).

Those methods of treatment are of great value which are directed to the toning up of the body in general and the nervous system in particular. The patient may go into the country or to the mountains or the seashore. Methodical treatment with cold water is good.

Frictions of the abdomen and entire body with cold water and alcoholic solutions are almost always attended by good results. On the other hand, those health resorts which often do good in the case of organic gastric disease are but seldom beneficial in nervous dyspepsia. Thus, we have frequently seen such patients, who had been sent by their physicians to Carlsbad, return worse rather than better. We have repeatedly found electricity valuable, although we surmise that its subjective effect may be of chief importance. Galvanism is applied along the spinal column, and also through the stomach horizontally, one large electrode being placed upon the epigastrium and the other on the back. It is well to reverse the current frequently. Faradization and massage of the abdominal walls are indicated, especially when there is constipation.

We need hardly add that the ætiological factors must not be overlooked. The patient must be warned against mental or bodily overexertion, emotional excitement, etc. The main point is to aim at a methodical moral training of the patient. He should learn to feel and to behave like a healthy person. He should regain his self-control, and not allow himself to be upset by every slight psychical shock, whether subjective or coming from without. It is self-evident that this goal is to be attained not by baths and prescriptions, but by proper moral guidance. The best proof of the correctness of our views, with regard to the true nature of this disease, lies in the therapeutic successes which can be obtained in this manner alone, although it is a pity that they are more often achieved by empirics and quacks than by scientifically educated physicians. It is therefore true of nervous dyspepsia, as of all similar neurasthenic conditions, that it is to be cured not by physic but by the physician.

SECTION V

Diseases of the Intestines

CHAPTER I

INTESTINAL CATARRH

(*Catarrhal Enteritis. Enteritis catarrhalis*)

Ætiology.—The majority of cases of intestinal catarrh, like gastric catarrh, are due to an abnormal irritation of the mucous membrane of the intestine by its contents. In many cases the irritants are of a mechanical or a chemical nature, and depend upon the quantity and quality of the food taken, which explains why catarrh of the stomach and catarrh of the intestine are so often combined with each other. Noxious substances are often introduced into the system by the ingestion of spoiled food, such as spoiled meat, fish, beer, etc.

To the intestinal catarrhs caused by improper food we may add the toxic catarrhs which are produced by the direct ingestion of poisonous substances into the digestive tract, or by the imprudent use of certain drugs, especially active cathartics.

A great many cases of intestinal catarrh are due to infectious influences; these include most of the apparently spontaneous catarrhs, and also many, if not all, of the catarrhs attributed to taking cold or getting wet, and, finally, those affections which often develop epidemically or endemically in hot weather, and which we term summer complaint, cholera morbus, etc. Cholera morbus is an especially severe form, and it will be described more fully later on. We must also mention here that intestinal catarrh is often one symptom of other general infectious diseases, such as typhoid, dysentery, septic diseases, or severe malaria.

In a final class of cases intestinal catarrh develops from disturbances of the circulation, which cause a passive hyperemia of the intestinal mucous membrane. Diseases of the liver and portal vein, and also chronic diseases of the heart, kidneys, and lungs, are the chief affections which produce a stasis in the portal system, and thus an intestinal catarrh; but here the stasis is probably, in most cases, only a predisposing factor in the development of the catarrh, since the action of all other irritants is made easier by the disturbance of the circulation.

The frequency of intestinal catarrh in both sexes, and at every age, is well known. Children, above all, have a pronounced tendency to diseases of the intestine, so that, by a probable estimate, almost one third of the illnesses of children are to be referred to the intestinal canal. We will give a special description of intestinal catarrh in children on account of this fact.

Pathological Anatomy.—The pathological changes in catarrhal inflammation of the intestines are essentially the same as are met with in the inflammation of any other mucous membrane. Redness and swelling of the mucous coat, increased secretion of mucus, and in severe cases purulent products on the surface of the membrane, and a cellular infiltration of the tissue itself,

are the well-known processes characteristic of all catarrhal inflammations. The solitary and agminated follicles often swell in follicular catarrh, and they may finally become the seat of superficial follicular ulcers. We often find, especially in children, superficial erosions on the rest of the mucous membrane, and in severe cases the so-called catarrhal ulcers.

If the catarrh has lasted a long time, we sometimes find a considerable thickening of the mucous membrane, which is due to hyperplasia of the connective tissue, and gives an uneven, puffy appearance to the internal surface of the intestine. Circumscribed hyperplasia of the connective tissue may actually lead to the formation of polypi. If the orifices of Lieberkühn's follicles are stopped, we have a cystic degeneration of the follicles from the retention of the intestinal juice.

We very often find, however, a considerable atrophy of the mucous membrane, especially in the chronic intestinal catarrh of children. This atrophy, which has been carefully investigated, especially by Nothnagel, affects chiefly the glandular layer of the mucous coat. In place of the glands, which in many parts may wholly disappear, we find connective tissue more or less rich in cells. The atrophy is usually most pronounced in the colon and the lower part of the ileum. The muscular coat may also take part in the atrophy.

Certain peculiarities of catarrh affecting single portions of the intestines will be mentioned later on.

Symptomatology.—The symptom by which chiefly we determine an affection of the intestinal canal, and which in the milder cases is often almost the only sign of an intestinal catarrh, is diarrhea—that is, abnormally frequent stools of a looser consistence than usual; yet, strictly speaking, we should not attribute every diarrhea to a catarrh of the intestinal mucous membrane, since a large number of influences may directly produce an increased peristalsis and a consequent diarrhea. Thus, for instance, it is a well-known fact that sudden terror or great anxiety may sometimes cause an obstinate diarrhea in a very short time. In nervous and neurasthenic conditions, we sometimes have a chronic diarrhea which can be due only to abnormal processes of innervation—"nervous diarrhea." The diarrhea which may arise immediately after taking cold is also merely the result of abnormally great peristaltic movements excited in a reflex manner. Probably a number of chemical and infectious irritants may also stimulate the movements of the intestines, and thus set up a diarrhea, without causing at the same time a catarrh of the mucous membrane. Practically, however, we cannot make a sharp distinction between diarrhea and intestinal catarrh; and, in most of the diarrheas which have lasted for some time, we are certainly right in supposing that there are actual anatomical lesions of the intestine as well as functional disturbances.

There are two chief factors which cause diarrhea in intestinal catarrh. In the first place, as has already been intimated, the same injurious substances which cause the catarrh also excite peristalsis. The many products of the abnormal processes of decomposition in the intestine also exert a like influence. Besides the abnormal irritants, however, we ought also to consider an abnormally great irritability of the diseased intestinal walls in catarrh. Thus it happens that the fluid contents of the intestine are expelled by the vigorous peristaltic movements (which the patient himself often feels as a "rumbling in the abdomen") before the normal consolidation of the feces is completed

by the absorption of water. The food, under normal conditions, passes through the small intestine in two or three hours, and thus the consolidation of the feces takes place, as is well known, almost exclusively in the colon. We see, therefore, why the diarrhea owes its origin chiefly to the increased peristalsis of the large intestine, although in many cases the peristaltic action of the small intestine is also increased. Besides increased peristalsis, another circumstance, in certain cases, contributes to the diarrhea, viz., the greater fluidity of the contents of the intestine due to the increased secretion of mucus and the exudation caused by the catarrhal inflammation.

In some intestinal catarrhs, especially those due to congestion, the diminished absorption of water from the intestines in consequence of the circulatory disturbance must be taken into account as a possible factor in addition to the increased peristalsis.

The diarrheal dejections show a considerable difference in regard to their minor characteristics. Their number varies very much. There are sometimes two or three, and sometimes ten or more, evacuations in the twenty-four hours. The consistence of the stools is paplike, or almost wholly watery. This is due to the abnormal amount of water in them, amounting to ninety or ninety-five per cent, while the amount in normal stools is about seventy-five per cent. The color of the thin stools in intestinal catarrh is usually bright yellow, but they are sometimes greenish from the admixture of bile pigment, and sometimes slimy (*vide infra*).

The odor of the dejections is frequently extremely foul; in other cases (with very watery movements) it is comparatively insignificant. In only a part of the cases does microscopic examination give us information as to the extent and intensity of the catarrh. We usually find the remains of the food, muscular fibers, starch granules, and fat, and also countless bacteria, and often triple phosphates, occasional blood and pus corpuscles, and cylindrical epithelium. Further peculiarities will be mentioned below.

Besides the diarrhea, there is often, but by no means always, abdominal pain in intestinal catarrh, either continuous or having the character of paroxysmal, so-called colicky pains. In catarrh of the rectum there is that constant painful desire to go to stool which we term tenesmus.

Physical examination of the abdomen gives, on the whole, few results. Sometimes the abdomen is flat, and sometimes there is meteorism. Marked peristaltic action of the intestines often causes gurgling and rumbling noises—*borborygmi*. On palpation, the abdomen is often sensitive. The peculiar colicky pains, however, are, as a rule, alleviated by external pressure. In rare cases we may detect a fluctuation on palpation if the intestine contains much fluid. The results of percussion depend largely upon the contents of the intestines. There is dullness on percussion if the intestines are full, and also if they are contracted and devoid of air.

In many cases of simple diarrhea the general health is practically unaffected, but in other cases of acute intestinal catarrh, especially in the severe infectious forms, the disturbance of the general health may be considerable. The patient feels so dull and weak that he stays in bed. We often see a moderate rise of temperature, between 100° and 102° F. (38° to 39° C.). There are very often gastric symptoms also, especially loss of appetite, coated tongue, and vomiting. Other organs are quite rarely affected, except in

duodenal catarrh, when the liver is involved (*vide infra*). In acute infectious intestinal catarrhs there is sometimes an eruption of herpes on the lips. We have repeatedly seen, in severe cases of acute enteritis, marked muscular and articular pains, and even slight but manifest swelling of the joints. There may also be albuminuria, casts, and even the signs of acute nephritis, as a sequel of enteritis.

Different Forms of Intestinal Catarrh.—Since the intestine is an organ which is only slightly accessible to physical examination during life, and since we can only rarely make a post-mortem examination in the mild diseases of the intestine, our knowledge as to the different forms of enteritis is defective in many respects. In practice we content ourselves in most cases with diagnosticating an intestinal catarrh simply from the existence of diarrhea, without laying much stress upon the special variety; but in many cases some points can be obtained which give more accurate information as to the seat of the catarrh. The distinction between acute and chronic intestinal catarrh is also of practical significance.

Duodenal catarrh can be diagnosticated only if it is combined with jaundice. The details regarding it may be found in the chapter on catarrhal jaundice.

Isolated catarrh of the small intestines, of the jejunum and ileum, is probably only of rare occurrence, except when the upper portions of the colon are involved. We can very rarely diagnosticate it with certainty, but there are a number of factors which permit us to decide that the small intestine is chiefly affected, or at least that it is involved in the disease. In the first place, we may assume an affection of the small intestine, for obvious reasons, in all those cases in which there are also gastric disturbances. It is evident that, in the frequent combination of gastric and intestinal catarrh, the portions of the intestine nearest the stomach will be chiefly affected. Physical examination of the abdomen also gives some indications, since the slight sensitiveness and swelling of the abdomen, as well as the visible abnormal peristaltic action, affect chiefly the middle and lower portions of the abdomen in catarrh of the small intestines, while the analogous symptoms in catarrh of the large intestine affect the lateral and upper portions of the abdomen, corresponding to the anatomical course of the colon. We cannot make a sharp distinction, however, in this respect. The results which auscultation and percussion over the abdomen give in regard to the point of origin of the gurgling sounds and the fullness of the loops of intestine are very rarely unequivocal.

Careful examination of the stools gives us more information. As has already been said, we need not have diarrhea in a catarrh confined to the small intestines, since diarrhea is due only to the increased peristalsis of the large intestine; hence, diarrhea is absent, for example, in most cases of duodenal catarrh (catarrhal jaundice). In more extensive catarrh of the small intestines the firm stools passed may, however, be regarded as pathological, because, on microscopic examination, they appear intimately mixed with little lumps of hyaline mucus (Nothnagel). As a rule, of course, catarrh of the small intestines is combined with a catarrh of the upper portion of the large intestine. Then we have a diarrhea, but the thin stools show certain peculiarities which point to an implication of the small intestines. As a result of the

increased peristalsis of the small intestines, we find certain constituents in the stools which are normally contained in the small intestines, but which under normal conditions are no longer to be met with in the feces in the large intestine. We find here, in the first place, undigested constituents of the food (so-called lientery), large masses of muscular fiber, or even fragments of meat which may be recognized by the naked eye, and also starch and fat. Of course the opposite hypothesis does not hold good, that, if we find a large amount of the undigested portions of the food in the stools, it must necessarily always point to a catarrh of the small intestines, since the digestion may be impaired by other circumstances, and increased peristalsis of the intestines, from any cause, must result in the same symptoms.

Recently, much attention has been devoted to the careful microscopical examination of the stools (A. Schmidt, Strassburger, and others). If the patient receives a fixed test diet of meat, fat, and carbohydrates for several days, we can, by a microscopical examination of the feces, determine which class of food stuffs show a particularly marked disturbance in their absorption. These examinations, which are interesting but difficult of interpretation, have not been as yet extensively employed in general practice.

If the stools contain bile in addition to some portions of the food, it is to a certain degree characteristic of catarrh of the small intestines. Under normal conditions the contents of the small intestines alone show Gmelin's test for bile pigment, while the contents of the large intestine, and also the normal stools, do not. In intestinal catarrh, with increased peristalsis of the small and large intestines, there is, however, often quite a large admixture of still undecomposed bile pigment. The green stools which are so often seen in the diarrhea of children, and more rarely in that of adults, are also well known. Such stools usually show a marked color reaction with nitric acid. In other cases we find only certain constituents of the stools stained with bile—a fact to which Nothnagel has called special attention. Yellow pigmented bits of mucus, and cylindrical epithelium and round cells stained with bile, are especially characteristic of the diarrhea of catarrh of the small intestines.

Catarrh of the large intestine is probably present in every diarrhea, as has been repeatedly stated, inasmuch as the thin stools can be explained only by an increased peristalsis of the large intestine; but in a number of cases we have symptoms which point especially to a disease of the large intestine, particularly of its lower portion.

Physical examination of the abdomen shows changes, such as swelling, sensitiveness to pressure, etc., chiefly in the lateral portions, corresponding to the course of the colon.

A marked tenderness to pressure in the region of the sigmoid flexure is of the greatest diagnostic importance. Undoubtedly, severe localized inflammation of the sigmoid flexure and different portions of the colon does occur, although the pathology of this affection, which is known as acute colitis and sigmoiditis, is still extremely obscure. Nevertheless, the diagnosis can occasionally be made with reasonable certainty, by careful palpation of the thickened and tender descending colon, in connection with the other symptoms (mucus in the stools, fever, etc.). In respect to ætiology, preceding constipation deserves especial consideration. The peritoneal coat of the colon is

occasionally involved in the inflammatory process (so-called pericolitis). Recently, such cases of acute febrile pericolitis have been repeatedly observed. In all inflammatory affections of the large intestine, a considerable admixture of macroscopically recognizable mucus in the stools is of diagnostic importance.

As we have seen above, the stools in catarrh of the small intestines also contain mucus, but it is intimately mixed with the other constituents of the feces, and hence it can usually be recognized only by the microscope. In catarrh of the large intestine, however, the mucus rather adheres to the outside of the other constituents, and is often present in large masses visible to the naked eye. If the catarrh affects the lower part of the large intestine chiefly, it may be that the intestinal contents are already formed into firm lumps, which may sometimes be wholly or partly inclosed in a layer of mucus. In acute catarrh of the lowest part of the large intestine the evacuations are sometimes composed chiefly of pure mucus, with a greater or less admixture of pus, as is seen especially in the "catarrhal flux" (see the chapter on Dysentery). The more the rectum is involved in the inflammation, the worse is that painful feeling of tension and pressure at the anus during and after the evacuation, which we term tenesmus.

Isolated inflammation of the rectum (proctitis) is, at least in part, directly accessible to examination by the finger or by the speculum. Painful tenesmus and an admixture of mucus, and especially of pus in the stools, are the chief symptoms of the disease. In most cases, however, we have to do, not with a primary disease, but with a secondary catarrh of the rectal mucous membrane, as a result of different morbid conditions in the vicinity of the rectum, or of new growths, syphilitic processes, etc., in the rectum itself. Periproctitis (ischiorectal abscess) belongs to the domain of surgery, and cannot be described here.

Intestinal catarrh is divided into an acute and a chronic form.

In the acute intestinal catarrhs, excluding the toxic inflammations, we class simple diarrhea, which usually passes off in a few days, and the severe enteritis, which is probably usually infectious, and is attended by a marked disturbance of the general health, by fever, and sometimes by gastric symptoms also, as well as by herpes, by occasional slight albuminuria, by articular pains, etc. It lasts from three to ten days. Furthermore, the different forms of acute catarrhal colitis described above belong to this category.

Cholera morbus (*vide infra*) is to be regarded as a special form of acute infectious inflammation of the gastric and intestinal mucous membranes.

Chronic intestinal catarrh either comes from an acute disease of the intestinal mucous membrane or gradually develops independently. In adults it is by no means a very common disease, at least as regards pronounced cases. It is observed with comparatively greatest frequency in drinkers, especially in habitually excessive beer drinkers. We personally know patients of this sort who have had four or five watery stools for many years. Moreover, chronic intestinal catarrhs occur occasionally in adults, as sequelæ to severe acute intestinal disease, particularly after typhoid, dysentery, malaria, etc. The most pronounced symptom of the disease is the disturbance of the bowel movements. In some cases there is continual diarrhea; in others, diarrhea alternates with obstinate constipation. With a careful mode of living,

the movements are frequently almost normal, but then even a trivial error in diet, catching cold, or psychical excitement (*vide supra*, nervous diarrhea) may suffice to produce a diarrhea. In respect to the character of the stools, we may refer to what has been said above. The demonstration of mucus in the stools is always the most important sign. The test-irrigation of the intestines, introduced by Boas, furnishes reliable information in this respect. After previous evacuation of the bowels, a rectal tube, connected to a large glass funnel by means of rubber tubing (as in gastric lavage, *vide supra*, page 509), is slowly introduced per rectum as far as possible into the large intestine, small quantities of lukewarm water being meanwhile allowed to flow in. The funnel is lowered as soon as a desire to defecate is evinced. The water flows back into the funnel, and frequently contains a distinct admixture of mucus, etc., which permits us positively to conclude that a catarrhal affection of the large intestine is present. The subjective symptoms of chronic intestinal catarrh consist of various unpleasant and painful sensations (pressure, borborygmi, colics), which appear particularly after errors in diet, and may become extremely disagreeable to the patient. In some cases the general nutrition suffers very little, while in others gradually marked emaciation and pronounced anæmia develop.

Treatment.—Most of the milder cases of acute intestinal catarrh need only a dietetic treatment. If the patient avoids all injurious substances for a few days, he recovers completely. The different gruels, such as barley and oatmeal gruel, and also weak broths, milk, and thoroughly toasted bread, or the German zwieback, are generally regarded as the most suitable food. The coarser vegetables and fruits, fat meat, and brown bread, are to be avoided as much as possible. The best beverage is tea, or claret diluted with water. In other respects we may refer to the dietetic rules laid down under the treatment of chronic gastric catarrh.

It is also an important rule, confirmed by much experience, to keep the abdomen warm. Children should always stay in bed, and adults should do so, at least in all severe cases. It is a good plan, particularly in children, to protect the abdomen from cold by a flannel band.

In many of the mild cases it is scarcely necessary to use internal remedies. Gum mixture (P. G.) or almond mixture is a good prescription if there is no other special indication, but in severe cases further medication may be proper. If we have reason to suspect some irritating ingesta or a collection of feces as a cause of the intestinal catarrh, a cathartic acts favorably at the beginning of the treatment in spite of the existence of diarrhea. Our best cathartic in such cases is castor oil or calomel. In all those cases in which many thin dejections point to a greatly increased peristalsis of the intestine, we use astringents, especially opium, which we give in the form of the simple tincture or the wine in doses of 10 to 15 drops, one to three times a day; or as a powder, 0.5 to 1 gr. (gm. 0.03 to 0.05) of opium with 1 gr. (gm. 0.05) of sugar, two or three times a day. It is also well to combine the opium with some mucilaginous vehicle, as 2 parts of laudanum to 150 of gum mixture or decoction of salep (P. G.), a tablespoonful every two or three hours. Tannic acid and the like are seldom employed in acute enteritis.

If there is severe colic, opium, or, under some circumstances, an injection of morphin is the best remedy. In milder cases it is sufficient to apply

warmth to the abdomen by warm poultices or hot towels. The colic, however, often depends upon the presence of old fecal masses in the intestine, when it is necessary to prescribe a cathartic, such as castor oil.

If there is painful tenesmus, it is usually relieved by suppositories of cacao butter containing extract of opium.

In chronic intestinal catarrh a careful regulation of the diet is of the greatest importance. To be avoided are fruits and sour or greasy dishes, indigestible vegetables and puddings, coarse bread, and, above all, beer; and to be recommended are tender lean meat (sirloin), sweetbread, fish that are not oily (such as trout and pike), potato *purée,* and particularly soft-boiled rice and sago. In regard to eggs and milk, individual experience must decide.

As beverages, warm or cold tea, cocoa, red wine and water, and particularly huckleberry wine, which we have used a great deal with good results, are to be recommended. Acorn cocoa is also frequently of value.

Medicinal treatment is directed chiefly against the chronic diarrhea. Next to opium, the astringents rank first, especially tannin, tannigen (acetyltannin) and tannalbin (tannin albuminate). The two last-named remedies have now almost entirely replaced tannin. They are employed in doses of 7 to 15 gr. (gm. 0.5 to 1) several times daily, either alone or in combination with opium. The older astringents, such as radix colombo, Lignum campechianum, catechu, etc., are now, perhaps undeservedly, only rarely prescribed. Among the metallic remedies, acetate of lead deserves a trial. The preparations of bismuth are frequently of decided benefit—viz., bismuth subnitrate, bismuth salicylate, and betanaphthol bismuth (orphol); these remedies are also frequently combined with opium (e. g., bismuthi salicyl., gr. v [gm. 0.3]; extr. opii, gr. ½ [gm. 0.02]; sacch. albi, gr. v [gm. 0.3], three powders daily). In some cases of chronic diarrhea the use of lime is recommended (calcium phosphate and carbonate, equal parts, a teaspoonful in a glass of plain or carbonated water three times daily).

In case the offensive odor of the discharges indicates abnormal decomposition in the intestinal canal, we employ naphthalin in doses of 1.5 to 5 gr. (gm. 0.1 to 0.3) several times a day. We often have to try various remedies in a particular case before we find one that is efficient. Great care should be exercised that there be no long periods of constipation; if necessary, we may use injections, saline laxatives, or castor oil.

In all cases in which the symptoms point to a serious affection of the large intestine, local medication can be employed.

We irrigate the large intestine daily with weak astringents, and sometimes with disinfectants. The necessary apparatus is very simple. It consists of a large glass funnel, to which a rubber tube, about half a meter long and with a proper tip, is attached. We may very well use, for an end piece to be introduced into the rectum, a long, soft, elastic œsophageal tube, which can easily be pushed quite high up. The fluids used for irrigation must always be warmed to about 85° F. (30° C.), and should be allowed to run in gradually and slowly. The amount of fluid used for one irrigation should be 2 to 3 pints (1 to 1.5 liter), or sometimes more. The patient keeps on his back during the irrigation. The knee-elbow position, which is much more uncomfortable than the dorsal, is only occasionally necessary. The fluids most used are a one- or two-per-cent solution of salicylic acid, solutions of salicylic and

boric acids combined, a one-per-cent tannin solution, or a solution of acetate of lead (1 to 1,000), diluted limewater, etc. Injections of bismuth (10 to 15 parts in 1,000 of water) are also occasionally employed with success.

Good results are often obtained in chronic intestinal catarrh by drinking the waters at Carlsbad, Tarasp, Kissingen, Marienbad, or Homburg, particularly in cases with occasional constipation.

It is of great practical importance to distinguish the genuine cases of chronic intestinal catarrh with an anatomical basis, from the common nervous disturbances of the digestive tract. Not infrequently the most diverse symptoms on the part of the intestine, including colic, irregularity of the bowels, etc., appear as part of a general neurasthenia or nervousness. As a rule, the nervous intestinal disturbances come on in attacks. The patients are very well at times, while at other times without any sufficient cause, or after slight colds, errors in diet, and even psychical excitement, very disagreeable symptoms occur, such as painful sensation in the abdomen, borborygmi, colics, sudden diarrhea, passage of mucus, etc. Evidently we are dealing with conditions of increased intestinal irritability which manifest themselves in some cases more in abnormal peristalsis, in others more in abnormal secretion. In most of such cases there is little benefit from internal remedies and strict diet, while appropriate general treatment with cold water, electricity, gymnastics, and massage is often attended with the best results. With regard to this, compare also the chapters on Nervous Dyspepsia and on Habitual Constipation.

APPENDIX

Membranous Enteritis and Mucous Colic.—The name of membranous enteritis or mucous colic is applied to a condition which is not very uncommon, and is therefore of practical importance. It is characterized chiefly by the habitual or periodic discharge with the feces of a great abundance of mucus, in the form of membranes. The disease is observed especially in women, but, in rare cases, also in men. It is noticeable that the patient is almost always a nervous, hysterical, or hypochondriacal individual.

In many cases a discharge of the membranes takes place in separate attacks, which occur either daily or at longer intervals, and are associated with severe colic ("mucous colic"). The membranes are gray or reddish gray, and often cylindrical or rolled up in a ball. They consist mainly of mucin—sometimes, it is said, of albuminoid matters also—and examined under a microscope they are usually found to contain an unusual amount of desquamated cylindrical epithelium, with a very small number of leucocytes, and perhaps also a few crystals of triple phosphate and cholesterin. In other cases there are scarcely any attacks of colic, but merely a persistent discharge of the above-described membranes and bits of mucus. The true discharges of the bowels are almost always very sluggish and very hard.

There is still much uncertainty as to the true nature of this diseased condition. Whether there is a genuine "enteritis" is doubtful, at least with regard to many cases. The noticeably frequent association of the disease with hysteria and neurasthenia seem to indicate nervous causes for the exaggerated production of mucus. The formation of the membranes probably takes place

through the rolling up of the mucus in the depths of the longitudinal folds of the colon during its cramp-like contractions.

The *course* of the disease is usually tedious, but complete recovery may occur; there is no danger. The nutrition of the patient may remain perfectly normal, but in some cases it is greatly impaired. This depends chiefly upon the character of the food taken and the other nervous symptoms present.

The *treatment* is partly local and partly, or rather chiefly, general, for the cure of the associated neurasthenia.

For local treatment, oil injections are to be particularly recommended (daily injections of about 500 c.c. warmed olive or poppy oil), or injections of weak solutions of bicarbonate of soda or greatly diluted limewater. The existing constipation should also be treated with a suitable, not too careful, diet, by massage and only in cases of absolute necessity with internal cathartics. For severe colicky pains we prescribe hot poultices and suppositories of opium or extract of belladonna. The very important general treatment consists principally in cold-water cures (half baths, rub downs, compresses), fresh-air treatment (seashore, mountains), electricity, etc.

Of late, severe cases of membranous colitis have been repeatedly treated, as have indeed severe inflammatory affections of the colon, by making an artificial anus. In this way the diseased mucous membrane of the colon is protected from irritation, and, moreover, drugs can be applied to it through the artificial opening. The author has been able to convince himself of the efficacy of this treatment in some very obstinate cases.

CHAPTER II

CHOLERA MORBUS

(Cholera nostras. Cholera infantum)

By the name "cholera morbus" we mean an acute disease of the stomach and intestinal canal of a definite form, whose symptoms in severe cases greatly resemble those of genuine Asiatic cholera. It is in the highest degree probable, from the whole course of the disease, that cholera morbus also depends upon an acute infection of the body by a specific germ; but this germ has not yet been identified.

Cholera morbus comes on usually as an epidemic, and almost exclusively in the hot summer months—June to August. Hence it is often termed summer cholera. Children in the first two years of life are chiefly attacked, especially those who are artificially fed or who have recently been weaned. The disease also attacks older children and adults, but much more rarely.

The symptoms of cholera morbus are those of a severe acute gastroenteritis. The disease begins suddenly, or after some slight warning, with violent vomiting and severe diarrhea. In some cases one of these symptoms predominates, and in others the other. The vomitus consists partly of the food taken, and partly of a slimy, watery substance. The stools at first retain their fecal character, but they soon become more colorless and more watery, so that they sometimes approach the well-known rice-water appearance of the stools in gen-

uine cholera. Abdominal pain is usually absent, but a feeling of pressure and constraint in the epigastrium is often present. The diminished secretion of urine and the frequent muscular pains cause the whole type of the disease to resemble genuine cholera still more closely. There is sometimes a cutaneous eruption resembling roseola.

The severe constitutional disturbance is especially characteristic. The patient becomes extremely dull and has a wasted look, the voice is weak and hoarse, an unquenchable thirst sets in, the pulse is very small, the skin of the face and the extremities is cool and livid; in short, we have the pronounced picture of a general collapse. The body heat also falls, although at the first stage of the disease there is often a rise of temperature.

[The temperature is always high, even during the stage of collapse, when the skin and extremities are cool to the touch; if the thermometer is introduced into the rectum—generally the best place, by the way, to take the temperature in young children—it will rise to 101° to 102° F., and is more apt to reach 104° to 107° F. This shows that inflammation plays a large part in the pathology of the disease.]

The picture of a severe general disease is especially prominent in cholera infantum. In severe cases of this form of the disease the general restlessness, which at first exists, rapidly passes into somnolence. The child lies with sunken, half-closed eyes, the conjunctivæ are slightly injected, the corneæ are cloudy, the face is pale and cyanotic, the fontanelles are depressed, the skin is cool, and the pulse is small and frequent and it can scarcely be counted. Amidst these symptoms, death comes on in coma or with slight convulsions. There can scarcely remain room for doubt that these severe cases of gastro-enteritis are of infectious origin, and that the constitutional symptoms are the result of toxic matters generated in the intestine under the influence of the microörganisms. Another peculiar symptom which occurs in severe cholera infantum is the so-called sclerema adiposum. While the temperature keeps sinking the extremities become peculiarly stiff and rigid, and the skin grows pale and firm. This phenomenon is caused by the fat in the fat cells of the subcutaneous connective tissue stiffening as a result of the low [superficial] temperature.

The mortality of children with cholera infantum is very marked, especially in large cities, and among the poorer classes of society. Severe cases usually end fatally in a few days, but, on the other hand, many cases recover, either because the course of the disease from the first is not so severe, or because cases apparently hopeless take a favorable turn. In adults it is extremely rare to see cholera morbus terminate unfavorably. Patients also recover quite rapidly from apparently severe conditions, although the stomach and intestines often remain rather sensitive for a long time.

The anatomical appearances in children who die of cholera infantum usually contrast, from their insignificance, with the severe symptoms observed during life. The catarrhal affection of the gastric and intestinal mucous membranes is not at all prominent in the cadaver, and the solitary follicles and Peyer's patches show only a slight swelling. A careful microscopic examination of the intestinal mucous membrane shows, however, quite severe inflammation. The other lesions which are most frequently seen are lobular atelectases in the lungs, venous hyperæmia and œdema of the pia mater, and slight lesions of the kidneys.

The diagnosis of cholera morbus presents no difficulty if the characteristic symptoms of the disease are present. The distinction between it and genuine Asiatic cholera used to be occasionally quite difficult, and it was rendered possible only by considering the ætiological factors, and the evident connection between the individual case and other cases of undoubted cholera. By Koch's discovery of the comma bacillus in Asiatic cholera the distinction between the two diseases has now become absolutely certain. In all suspicious cases, therefore, we must examine the dejections for comma bacilli, and upon the result of this examination depends the determination of the proper means of prophylaxis.

The treatment of cholera morbus in adults must be first to take special care to limit the diet. The food should be only gruels, or at most broth and milk. It is a good plan to give the milk iced, and in small amounts. The distressing thirst is best relieved by cracked ice or cold tea. Wine (iced champagne) is to be given if the general weakness becomes marked.

Among drugs, opium is the most effective remedy, and, whether in powder, as the extract, or in liquid form, as laudanum, it is the first thing to use to relieve the diarrhea and vomiting. All other remedies, such as nitrate of silver, are quite subordinate to opium. We may combine small doses of calomel with the opium. If there is a severe collapse which threatens life, it is advisable to make a subcutaneous injection of water containing 0.6 per cent of salt.

In the case of children, it is best to discontinue milk entirely for several days, or, at the most, to give ice-cold milk by the teaspoonful. A supply of fluids is alone essential, either in the shape of boiled water or weak tea (chamomile tea). Mucilaginous drinks (thin gruels) and albumin water must also be used only with care.

We should rather hesitate in prescribing opiates, although here small doses of opium, 1 or 2 drops of laudanum according to the age of the child, may often be indispensable. In fresh cases calomel has obtained a great reputation, a sixth of a grain (gm. 0.01) two or three times a day. If nothing will stay on the stomach, we may try a subcutaneous injection of 0.6 per cent saline solution. If there are symptoms of severe collapse, we should immediately employ hot baths at 100° F. (30° R.), with the addition of 10 to 15 ounces of mustard (gm. 300 to 500) and hot packs, as well as such stimulants as camphor or alcohol subcutaneously. To guard against desiccation of the sclera and cornea, the eyes should be covered with compresses moistened with solutions of acetate of lead, corrosive sublimate, etc.

CHAPTER III

INTESTINAL CATARRH OF CHILDREN

(Pedatrophy. Acute and Chronic Intestinal Disturbances of Children)

The great frequency and the practical importance of the "dyspeptic conditions" in children in the first years of life, which conditions are associated with severe disturbances of nutrition, justify a short description of them, but we must refer to the special manuals on children's diseases for a detailed account.

Ætiology and Pathological Anatomy.—That diseases of the digestive organs play so large a part in children's troubles is owing, on the one hand, to the great sensitiveness which the infantile organism, and particularly its digestive apparatus, shows to the irritants which are brought in contact with them, and, on the other, to the too common mistakes which the child's parents and nurses show in its feeding. Of course it is not always ignorance and neglect, but often, unfortunately, poverty and want which cause children to suffer, and explain the terrible mortality in the first years of life.

The simple fact that by far the larger number of children who suffer from dyspeptic and atrophic conditions are fed artificially, leads us to the belief that the cause of most intestinal diseases of children is to be sought for in a defective and insufficient dietary.

It has been determined statistically that the mortality of bottle-fed infants is approximately twenty times greater than that of breast-fed. For a time there was a tendency to ascribe the detrimental influence of artificial feeding principally to impurity of the milk, i. e., to infectious causes. Flügge, Czerny, and others have, however, shown that the use of sterilized food has no marked influence on infant mortality. Though the deleterious action of bacteria (*vide infra*) can by no means be entirely disregarded, the chemical and biological differences between mother's milk and all artificial foods, and, above all, the quantitative differences in the composition of the different artificial foods, furnish in themselves a number of causes for disease. These noxious causes and their sequelæ have been studied in detail for the different classes of artificial foods (Czerny). The harmful action of cow's milk is due, perhaps, partly to the chemical peculiarities of its proteids (*vide infra*), partly to its distinctive enzymes, and partly to an admixture of noxious substances derived from the fodder eaten by the cows, etc. In the first place, the "overfeeding" of infants with proteids, and, above all, the continuous overabundant supply of fats, play a very important rôle in the "milk-diet disorders." Another class of nutritive disorders is to be ascribed to the very common use of gruels (*Mehlsuppen*) and similar preparations as food for infants (so-called farinaceous-diet disorders). Here both the continued lack of fats and also an insufficient supply of proteids and salts play a rôle. To what extent the too early administration of eggs and meat can produce protcid-diet disorders, due to the specific action of "hetero-albumins," has not yet been determined with certainty. The assumption of some pediatrists, that the proteid of cow's milk—i. e., casein—is particularly indigestible and harmful, has not been conclusively proved. In isolated cases, again, nutritive disorders due to gelatin have occurred from the continued administration of strongly gelatinous broths prepared from calves' bones.

Besides incorrect feeding, in regard simply to quality or quantity, infectious influences, as already stated, also play a great part. Pathogenic bacteria are occasionally introduced as such into the stomach and intestines with the food. Or, we are dealing with bacteria normally present in the digestive tract which, however, under peculiar conditions have increased in numbers and possibly also in virulence. In spite of many investigations, it has not been conclusively determined which particular group of bacteria is to be considered the principal cause of the dyspeptic conditions in children. In many cases virulent types of the bacillus coli appear to play the chief rôle; in other

cases, streptococci, diplococci, etc. The infectious nature of the disease has a relation to the fact that dyspeptic conditions are much more frequent during the summer than the winter months. It should be remarked, however, that the deleterious action of the bacteria must be frequently regarded, not as a true infection, but rather as an intoxication, since the food becomes decomposed by bacteria either before being administered or afterwards in the digestive tract. In this way a number of toxic substances are produced ("alimentary toxicosis," according to Czerny and Keller). It is mainly the acids produced by decomposition of the milk sugar and the fats (lactic acid, fatty acids, etc.), but occasionally also the fermentation products of the farinaceous carbohydrates, which cause diarrhea, increased production of mucus in the intestines and general toxic disorders. Some of the clinical pictures have a strong resemblance to acid intoxications (acidosis, as in diabetic coma). Particular attention should also be paid to the loss of water from which the body suffers.

Symptomatology.—With this conception of the symptomatology of the disease as the expression of a bacterial intoxication, the strikingly insignificant anatomical changes found at the autopsies of children who have died of "intestinal catarrh" entirely coincide. What were formerly considered to be signs of organic intestinal disease have been shown in many instances to be merely post-mortem changes. Nevertheless, the swelling of the follicles (follicular catarrh) is occasionally striking. Follicular ulcers also occur. In other cases an atrophy of the mucous membrane is the main change. Chronic thickening and redundancy of the mucous membrane are of rarer occurrence. In most of the severe cases the large intestine, and also the lower portion of the ileum, are the chief seat of the changes. We often find a swelling of the mesenteric lymph-glands, and also a fatty liver. In the lungs extensive atelectases or nodules of catarrhal pneumonia often develop as a result of the imperfect respiration. Other not infrequent complications resulting from the general infection or intoxication are otitis, nephritis, cystitis, etc. The symptoms of the dyspeptic disturbances are, in the first place, those due to the intestinal trouble, and, secondly, the quite rapid disturbance of the child's general nutrition.

The condition of the stools is the most important intestinal symptom. The normal dejection in children until they are weaned is of the color of the yolk of an egg, of a rather pasty consistence, and of a faintly sour smell. In most intestinal disturbances the stools are more frequent, six or seven, and even more, a day. The stools are not of an even consistence, but contain flocculent and lumpy masses, which look as if they had been chopped up. Or, they are thinner, more watery, contain large flakes and lumps (undigested bits of casein and other remains of the food), as well as a marked admixture of mucus, either as "sago grains" or larger glassy masses. The odor of the stools becomes fetid, the reaction is frequently alkaline, the color throughout or in part is frequently distinctly green. This green discoloration probably depends principally on a decomposition of the bile pigments. Whether bacteria also play any rôle in this process is still doubtful. Sometimes the stools do not acquire the green color until after they have been passed. With the microscope we find in severe cases, besides particles of food, leucocytes and epithelial cells, threads and clumps of mucus, and innumerable bacteria of

all shapes. There are also crystals of triple phosphates, when the stools have an alkaline reaction, and needle-shaped crystals of the fatty acids, as well as plates of cholesterin, when the discharges are acid. Small quantities of blood are also occasionally found in the stools, which naturally always points to more marked anatomical changes in the intestinal wall. Certain forms of enteritis are characterized by exceedingly imperfect absorption of fats. The stools are then light yellow or greenish yellow, and of a thin consistence; they smell strongly of fatty acids, and contain a very plentiful amount of undigested fats (so-called "fatty diarrhea").

There is no definite distinction in regard to the dejections in catarrh of the large and of the small intestines. On the whole, the rule holds that, in catarrh of the small intestines chiefly, the stools are larger, they are passed with more wind or gas, and show a more uniform consistence; while in catarrh of the large intestine they are smaller but more frequent, ten or twenty a day, are passed noiselessly, are associated with tenesmus, and show a different consistence in their various parts, partly normal, partly thin, partly slimy, etc. Examination of the abdomen reveals, as a rule, in catarrh of the small intestine that the abdomen is much swollen, while in catarrh of the large intestine it is often deeply sunken. The tissues about the anus are often red and sore and the mucous membrane prolapsed.

The liver is often enlarged and distinctly palpable, and sometimes the spleen also. The swollen mesenteric glands can be felt in isolated cases. We often find disturbances in the stomach, vomiting, eructations, etc., as well as trouble in the intestines. There may be thrush in the mouth, or the development of aphthous ulcers. Among other complications, we see diseases of the respiratory tract, including bronchitis, atelectasis, and catarrhal pneumonia; also albuminuria, eczema, furunculosis, otitis, nervous disturbances, etc. Rachitic and tuberculous changes in the lungs, intestines, and lymph-glands may be combined with the intestinal catarrh.

In almost all long-continued cases, however, the general disturbance of nutrition, the atrophy ("pedatrophy," "athrepsia") of the child, takes the foreground in the picture of the disease. Accurate observation of the body weight through weekly or biweekly weighings is imperative in order to judge the course of the disease. In severe cases of chronic enteritis, or those that have been treated improperly, the atrophy finally attains its maximum intensity. The muscles become shriveled and flabby, and the whole body finally becomes so much emaciated that the pale, dry skin hangs in broad folds and wrinkles about the bones, whose prominences are everywhere visible. The face is sharp, and has an aged expression ("Voltaire-face") from the many little folds of the skin. The eyes are dull, lusterless, and wide open; the voice is merely a low, hoarse whimper. The abdomen is deeply sunken, or in some cases it is swollen by meteorism, in peculiar contrast to the emaciation elsewhere, and its surface is traversed by bluish veins.

Treatment.—If we would give a full account of the treatment of the atrophic conditions in children due to digestive disturbances, we must include in our consideration the entire hygiene and care of children in health and disease, for all children's physicians are united in the opinion that, as the cause of most intestinal diseases in children is to be found in improper feeding, so recovery from existing digestive disturbances can take place primarily only

by a corresponding proper and judicious feeding. In what follows we can refer only to the most important principles and general points which are here to be considered.

The only proper and natural food for a child in its first year is breast milk. All dyspeptic conditions are much rarer in children who are nursed than in bottle-fed children, and, when they do occur in children at the breast, they often are only of brief duration. They are then to be referred usually to certain disturbances in the mother, such as disease, improper food, or severe mental excitement. The return of menstruation or a new pregnancy has sometimes an unfavorable influence on the character of the milk. It is very important to see that the breast is given at regular intervals to the infant. Generally the feedings should be given every three hours during the day, so that the number of feedings in twenty-four hours approximate six or seven. The duration of each nursing is approximately ten minutes, and should not exceed twenty. Under normal conditions the increase in weight for the first quarter of the first year is about 6 to 7 ounces (gm. 180 to 200) weekly, for the second quarter about 4 ounces (gm. 120), and for the third 3.5 ounces (gm. 100). By weighing the infant before and after nursing we can find out whether the child receives a sufficient quantity of food. The quantity of milk taken at each feeding should be approximately 2 or 3 ounces (gm. 50 to 80) during the first quarter of the year, 3 to 4 ounces (gm. 80 to 120) during the second, still more later on. If the secretion of milk in the nursing woman diminishes, or is insufficient from the outset, we can give diluted cow's milk (*vide infra*) in addition to the breast milk. Weaning of breast-fed children occurs gradually during the seventh to the ninth month. Cow's milk, weak broths, soft-boiled eggs, etc., are given in the transition to ordinary food.

In selecting a wet nurse the general nutrition and health (tuberculosis, syphilis), as well as the character of the breasts and nipples, must be considered. If possible, the child of the nurse should also be examined; its age at the time of taking the nurse should be between one and three months. The food of the mother or wet nurse must be abundant, excluding, however, all indigestible, strongly spiced and acid foods. Attention should be paid to a sufficient but not overabundant supply of fluids (milk, cocoa, beer, as much as a quart daily). It is of the greatest importance to get a wet nurse in those cases where the child does not thrive on artificial food and continually has dyspeptic troubles. Feeding by the milk of a wet nurse is then the only remedy which, at least in many cases, by saving the child's life, repays the many annoyances and quite large expense which a wet nurse causes. Complete, and sometimes even quite rapid, recovery may often be obtained through a wet nurse, even in cases of severe chronic intestinal catarrh, when atrophy and weakness are already very far advanced.

If the mother is unable to nurse the child, and it is impractical to get a wet nurse, the question of artificial feeding of the infant must be considered. The best substitute for mother's milk is cow's milk. It should be obtained from healthy cows, which have been fed on hay (dry fodder) and not on fresh grass, distiller's mash, etc. General cleanliness is of the greatest importance in the process of collecting the milk. The milk must reach the consumer in the shortest possible time, and then be boiled immediately for purposes of sterilization. Soxhlet's sterilizer is well suited to this purpose,

and is consequently much used. As cow's milk contains a considerably greater percentage of proteids and, on the other hand, a smaller percentage of fats and sugar than mother's milk, it is never given pure to children, but in diluted form, and, if necessary, even with certain additions. As a general rule, one part of the milk must be diluted, according to its quality, with two or three parts of boiled water in the first months, in children from four to six months old with equal parts of water, and in older children with about half as much water. At the age of about nine to twelve months, the child may have undiluted milk. In general we give the milk warmed to about 95° F. (35° C.), but children with gastro-intestinal catarrh often bear cold milk, given in small amounts, better than warm. Since the percentage of fats and milk sugar becomes decidedly too low by dilution, many attempts have been made to obviate this difficulty by special additions and methods of preparation. The addition of milk sugar is the most common. Heubner recommends a mixture of equal parts of cow's milk and a six-per-cent solution of milk sugar, whereas Soxhlet attempts also to replace the fat deficit with sugar, and consequently uses a still greater addition of milk sugar. Other pediatrists (Biedert, etc.) add fat in the shape of cream to cow's milk. By centrifuging milk diluted to half strength and adding milk sugar, the attempt has also been made to prepare a milk richer in fats and more closely resembling mother's milk (Gärtner's fat milk, etc.). Finally, preserved cream and vegetable fats (Lahmann's "vegetable milk") have been recommended as additions. In raising children on cow's milk, also, a regular division of the feedings is absolutely necessary. The interval between the feedings must be a three-hour one in the day and at night from six to eight hours. The quantity of milk taken at each feeding is slightly larger in bottle-fed than in breast-fed infants.

If pure cow's milk cannot be obtained or is not well borne, the artificial foods (consisting generally of milk and dextrinized flour), which are prepared in great number by the modern industrial chemical firms, must then be considered. In spite of the fact that all artificial foods have their great drawbacks, and should therefore be regarded solely as emergency foods, their use, under certain conditions, cannot be entirely obviated in practice. It cannot be denied that in certain cases good results are obtained with artificial foods. Among the "infant flours" which have been used with especial frequency, we may mention the preparations of Nestlé, Kufeke, Löfflund, Theinhardt, Mellin, etc.

As soon as disturbances in the functions of the stomach and intestines, as well as interference with the regular increase in weight and impairment of the general condition of the child occur, the treatment, in the first place, must always be a dietetic one. The physician should always first ascertain if the feeding of the child has been conducted in a correct and careful manner, and whether the simple observance of the previously expressed principles will not in themselves suffice to correct the disturbance. Where we are dealing with artificially fed children, we must first see whether the percentage composition of the food is not sufficient in itself to explain the disturbance, and, if so, make our changes accordingly. The "milk-diet disorders"—characterized by an insufficient increase in weight of the child despite a plentiful intake of milk, by a bad general condition, marked anæmia, a distended

abdomen, and gray, dry, foul-smelling stools—require a diminution in the daily quantity of milk, and the addition of mucilaginous and farinaceous decoctions (oatmeal, rice gruel, etc.). The "malt soups" can also be employed here with benefit. With nutritional disturbances due to farinaceous foods, the children appear at first to be well nourished. Later, a marked atrophy also develops, the children are very readily susceptible to secondary infections, occasionally they show a marked hypertonicity of the muscles. A discontinuance of the carbohydrates and a change to milk diet (preferably mother's milk, of course) can alone help here.

If severe acute digestive disturbances (vomiting, diarrhea) occur, it is best entirely to discontinue the milk and every other food for twenty-four hours. We give only some weak tea (chamomile tea) or albumen water (the albumen of one egg is beaten to a froth, mixed with one third of a quart of boiled water, strained, and sweetened with a little sugar). Even after an improvement in the symptoms has set in, the milk should be given diluted, and every four to five hours. If there is diarrhea, the dilution of milk with gruels is to be recommended (barley gruel, prepared by boiling a tablespoonful of barley with half to one quart of water for one hour, and then straining, oatmeal gruel, rice gruel, or decoction of salep, etc.). With older children, decoctions of acorn coffee may also be tried. In severe cases of acute gastro-enteritis with marked retching (especially where there is a suspicion of a toxic origin), gastric lavage has recently been repeatedly used with very good results even in young children. A Nélaton's catheter and plain water or a weak solution (0.01 to 0.2 per cent) of hydrochloric acid are used. Medication is entirely unnecessary in many cases of acute dyspepsia or acute enteritis. However, if the symptoms do not diminish after the requisite changes in diet, we must attempt to aid the treatment by internal medication. Calomel has won a specially wide reputation. It is given in doses of $\frac{1}{12}$ to $\frac{1}{6}$ of a grain (gm. 0.005 to 0.01) in powder. If the diarrhea lasts a long time, we may very well use opiates, although with great caution. The combination of calomel and opium often does good service.

℞ Calomel ..... gr. $\frac{1}{6}$ (gm. 0.01);
Extracti opii ..... gr. $\frac{1}{30}$ (" 0.002);
Pulv. acaciæ ..... gr. ss. (" 0.03).
M. et ft. pulv.

Sig.: One such powder, three or four times a day.

With little children we may put 2 to 4 drops of laudanum in 3 ounces (gm. 100) of liquid, such as gum mixture, salep decoction, muriatic-acid mixture, etc., and give a dessertspoonful of this every two or three hours. The tannic-acid preparations, tannigen and tannalbin (gr. jss. to v [gm. 0.1 to 0.3] per dose, several times a day), are also much used in infantile diarrheas, likewise bismuth subnitrate and bismuth salicylate (gr. ss. to jss. [gm. 0.03 to 0.1] per dose). For colicky pains we prescribe warm fermentations to the abdomen, embrocations of chloroform oil (massage), injections of chamomile tea, etc.

Many attempts have been made to check the abnormal processes of decomposition in the intestine by prescribing remedies which possess antiseptic

and antizymotic properties. Creosote has been warmly recommended by many 4 to 6 drops in 2 ounces (gm. 50) of water with half an ounce (gm. 15) of sirup, a teaspoonful every two hours. Other drugs used for the same purpose are naphthalin, one-half- to one-per-cent solution of dilute muriatic acid in water, and one-per-cent solution of chloral in water. The results, however, are doubtful. In chronic dyspeptic conditions of longer duration and in chronic intestinal catarrhs, as a matter of course, all the above-mentioned general dietetic regulations also must first of all be tried in the most careful and patient manner. The medicinal treatment, unfortunately, is mainly one of experimentation. The drugs used most frequently have already been mentioned—viz., tannigen, tannalbin, bismuth subnitrate, etc. It is to be noted that occasionally alkalies (particularly small doses of Carlsbad Mühlbrunnen) produce surprisingly good results even in young children. On the other hand, we have little confidence in efforts to arrest abnormal intestinal decomposition by the use of antiseptics. Nevertheless, we may occasionally try creosote as well as naphthalin and chloral hydrate (one part in one hundred), etc. When the stools are of a greenish color, lactic acid (a teaspoonful of a two-per-cent solution one quarter hour after each feeding) is said to act well.

If a large amount of mucus in the stools points to a catarrh of the large intestine, we may sometimes employ irrigation of the colon with excellent results. We inject the fluid, one-per-cent solution of tannin or alum, or solution of acetate of lead (1 to 3 to 1,000), once or twice a day. The amount of fluid to be introduced at once, by a Hegar's funnel, with a gum-elastic catheter, may reach one or two pints (half a liter to a liter).

With persisting gastric symptoms, repeated gastric lavage (*vide supra*) may be employed. The various symptomatic details (treatment of constipation, possible collapse, complications, etc.) need not be more fully considered in this place.

CHAPTER IV

TYPHLITIS AND PERITYPHLITIS

(Inflammation of the Appendix. Appendicitis)

Ætiology and Pathological Anatomy.—Inflammation of the cæcum and its vicinity has a special place among the diseases of single portions of the intestines. The reason why circumscribed inflammation so often develops here is to be found in the peculiar anatomical arrangement of the cæcum and its appendix, the vermiform process. It is evident that the conformation of the parts makes it particularly easy for inflammatory germs to settle in this narrow canal.

Formerly it was believed that the inflammation might often be limited to the cæcum itself. It was asserted that impacted fecal matter occasioned mechanical irritation of the intestinal wall, and thus facilitated the attack of chemical and infectious agents. In this way arose the doctrine of "typhlitis stercoralis," but such simple typhlitis has been demonstrated post-mortem only in isolated cases, and although it cannot be absolutely denied,

therefore, that under certain circumstances there may occur a genuine typhlitis, as a result of the accumulation of feces, yet its frequency has certainly been formerly much overestimated (Sahli). It is years since the author has made a diagnosis of "typhlitis stercoralis."

By far the greatest number of cases of acute inflammation in the ileocæcal region are perityphlitis; that is, inflammation of the peritoneum and the connective tissue surrounding the cæcum. From the point of view of pathological anatomy, we might term the inflammation of the retrocæcal tissue paratyphlitis, inasmuch as it is extraperitoneal, in distinction from the true intraperitoneal perityphlitis; but clinically we cannot maintain this distinction, or, at most, imperfectly. The starting point for perityphlitis is the vermiform appendix. To this there are rare exceptions in which the inflammation arises from perforation of the cæcum itself. The appendix, although of so little physiological importance and a merely rudimentary portion of the intestine, plays an important part in pathology.

The appendix has not been improperly compared with the crypts of the tonsil, in which likewise inflammatory agents readily find a nidus. The analogy is all the more appropriate, since the mucous membrane of the appendix is also particularly rich in lymphoid tissue (lymph follicles). The length of the appendix, its mode of insertion into the cæcum, and its position, are subject to many variations, which may also in part affect its susceptibility to disease. The appendicular artery, a branch of the cæcal artery, has no anastomosis with any other artery, is a so-called terminal artery, which is also a material fact in the causation of inflammation.

According to Sonnenburg and others, we distinguish a simple appendicitis and a gangrenous or perforative appendicitis. In simple appendicitis the inflammation is confined to the appendix itself. The mucous membrane is swollen and is beset with hemorrhages, the follicles are enlarged ("appendicitis granulosa"), the entire wall is infiltrated and rigid, and the serous coat is also generally markedly injected. In perforative appendicitis there is acute necrosis or gangrene of more or less extent, commonly at the tip of the appendix. In this way the inflammatory process extends to the surrounding tissues, and a perityphlitis develops from the appendicitis. The primary causes of the acute inflammation are still but little known to us. Occasionally there appears to be a special infectious factor. Thus, for example, appendicitis may apparently occur as a sequel to a preceding sore throat. At times a certain epidemic spread of the disease can also not be denied. In some cases slight traumata appear to be the exciting cause of the acute inflammation. Very frequently the acute attacks are merely exacerbations of a chronic inflammatory process, just as acute cholecystitis and biliary colic almost always originate on the basis of a chronic gallstone affection. This comparison is all the more apt, since the formation of concretions appears to play a rôle also in disease of the appendix. Even though present opinion is inclined not to ascribe as great significance to "fecal calculi" as was formerly done, it would, nevertheless, in our opinion, be incorrect to entirely deny their importance. Small fecal masses from the cæcum often enter the vermiform appendix, and, under some circumstances, they may remain there, more especially, perhaps, when the fold of mucous membrane (Gerlach's valve) at the orifice of the appendix prevents the return of the fecal masses

into the cæcum. The fluid in them is absorbed, they are very often incrusted with lime salts, and thus so-called "fecal calculi" are formed.

Foreign bodies, such as small fruit pits or seeds, etc., enter the vermiform appendix and perhaps give rise occasionally to the formation of a fecal calculus; but one must be cautious in assuming that any small, hard mass is a foreign body, for these calculi often have such a rounded shape that they were formerly considered, very erroneously, to be retained cherry stones, etc.

In many cases fecal calculi may remain in the vermiform appendix for a long time without producing any injurious results, but, as a rule, they cause a mechanical irritation of the mucous membrane which leads to inflammation, and often, in some circumscribed spots, to a pressure necrosis, and later to ulceration of the vermiform appendix.

If the ulcer of the mucous membrane cicatrizes, stricture formation, and occasionally hydrops (with suppurative inflammation, empyema), of the appendix occurs. As a rule, however, the inflammation extends to the adjacent tissues of the appendix. This extension of the inflammation is due in part to a migration of the bacteria and their toxins through the walls, or, more commonly (and this is the more important fact), to a perforation of the appendix. The mechanical pressure necrosis (anæmic decubitus and ulcer) is followed immediately by the entrance of the bacteria of inflammation and an extension of the inflammatory necrosis and inflammation. The pathogenic bacteria are not always the same. Virulent strains of the B. coli probably play the principal rôle, but occasionally pneumococci and other pyogenic organisms are found in perityphlitic abscesses.

All these processes may be very acute, or they may also take a fairly chronic course. Under certain conditions, the chronic process may light up into an acute exacerbation; this may frequently be repeated in the course of time. According to the acuteness and virulence of the process, we may merely find adhesions and agglutinations in the vicinity of the inflammatory focus, circumscribed serofibrinous exudates, a circumscribed abscess, or, in the most severe cases, a general diffuse peritonitis. In by far the greater number of cases agglutination and adhesions in the neighborhood of the appendix occur in time, so that the inflammation remains circumscribed. We designate this as a perityphlitis. In some of the milder cases there is only an infiltration of the tissues with pus cells, an inflammatory serous imbibition, perhaps also with a circumscribed fibrinous exudate. In the interior of the inflammatory tumor, which consists essentially of the thickened intestinal walls and the coils of intestine glued together, and perhaps sometimes also of impacted fecal matter, there is indeed often an actual abscess, small and localized, and lying usually in the immediate neighborhood of the appendix.

In all of the severer cases, however, large abscesses are formed. The various other possible pathological changes in perityphlitis will be best considered in connection with the clinical symptoms.

Perityphlitis is chiefly a disease of early life. Many cases occur in childhood (between five and twelve years), and again, more particularly between the ages of eighteen and thirty. Later on the disease becomes rarer. In our experience, the disease is also decidedly more common in the male than in the female sex. There appears to be a certain hereditary tendency to the disease in some families.

Symptoms and Clinical Course.—The symptoms of perityphlitis have usually a rather acute development. The patient has felt perfectly well, and, as a rule, has not even suffered from constipation, when he has a rather sudden pain in the ileocæcal region. There may be no reason for this, or there may be some such cause as stooping, lifting, or a long walk; in rarer cases, also trauma. Not infrequently there is slight vomiting, and the patient feels languid and feverish. The bowels may be constipated, but sometimes they continue regular. Many patients are obliged to go to bed at once, but others keep about for some days till they are forced to give up because of the aggravation of their symptoms, particularly the pain. It is a point of some practical importance that the pain is by no means invariably referred to the region of the cæcum at first, particularly as the position of the appendix is very variable. Not infrequently the pain is localized more in the middle of the abdomen or toward the left, or upward. It is not until subsequent days of the attack that the pain gradually settles in the true ileocæcal region.

The pains often radiate to the back, the region of the bladder, or the thighs. With severe pains respiration becomes superficial and costal in type, since deep diaphragmatic respiration increases the pains.

Upon objective examination we usually find distinct local changes in the ileocæcal region, even in the first days of the disease. Our manipulations should be extremely cautious. It may be possible by careful inspection alone to recognize a limited swelling. On palpation we are at once struck by the increased muscular rigidity of the abdomen on the affected side (also recognizable by a diminution of the abdominal reflex). In that case it is very important to determine whether tenderness is present. This may be very considerable or comparatively slight. The typical painful spot (McBurney's point) is said to be 1.5 to 2.5 inches (4 to 6 cm.) inward from the anterior superior spinous process, on the line joining this process with the navel. This localization does not possess great importance, because the painful spot varies in different cases. Sometimes the greatest pain is felt posteriorly more in the lumbar region, suggesting a retrocæcal paratyphlitic abscess (*vide supra*). Lastly, there is decisive importance in the discovery of abnormal resistance, or of a more or less sharply defined tumor due to the inflammatory exudation (*vide supra*). This tumor can usually be made out. For this purpose we recommend particularly the employment of the palm of the hand in gentle pushing movements, beginning on the left side and gradually proceeding toward the ileocæcal region. This is the best way of determining the size of the inflammatory mass. In milder cases it is also permissible to make deeper palpation, if we are cautious. The examination *per anum* is very important, especially in all suspicious cases in which the results of abdominal palpation are uncertain. We have repeatedly seen a number of cases in which no exudation could be demonstrated by external palpation, while a large inflammatory tumor could be distinctly felt through the rectum. The results of palpation are confirmed by percussion, which gives either a muffled tympanitic resonance or marked dullness over the seat of the disease. In general, however, the results of percussion should not be interpreted too strictly, for they are ambiguous, because of the variations in the degree of intestinal distention.

In addition to the local symptoms just described, careful attention should be paid to the general symptoms. Of great importance are the aspect and general condition of the patient. If the patient has no suggestion of the "abdominal facies" (*vide infra,* chapter on Peritonitis), it is very probable that the inflammation remains circumscribed. It is also important to study the chart of the temperature and pulse.

Some cases begin with a sudden fairly high elevation of temperature (up to 104° F. [40° C.]), ushered in by a chill; or the onset of the fever is slower, but the temperature remains moderately elevated (about 101° to 103° F. [38.5° to 39.5° C.]) for the next four or five days, and then subsides on the following days if the patient remains quiet. Defervescence occurs more or less rapidly by lysis, but frequently also in an almost critical manner. Not infrequently, after the first three or four days of the disease, a remarkable remission in all the symptoms occurs, and in the more favorable cases convalescence may then ensue. Very often, however, there is a recrudescence of the fever, and then great caution is urgently demanded. For renewed elevation of temperature after the fourth or fifth day of the disease always indicates an aggravation of the local process, particularly the onset of marked suppuration. The pulse ought to be watched as carefully as the temperature. The pulse rate is approximately from 80 to 100 at the onset. A lowered pulse rate is generally a favorable, and an increased an unfavorable, sign. A rapid, soft at times, slightly irregular pulse frequently indicates a beginning septic infection. Severe cases of septic peritonitis sometimes run their course with scarcely any temperature, but with a small, rapid pulse. [In such cases, the rectal temperature is often much higher than the oral.]

The bowels are almost always constipated from the start. Vomiting occurs frequently at the onset, but is absent entirely or nearly so in the lighter cases during the further course of the disease. Frequent eructations and a return of vomiting later on in the attack are unfavorable symptoms, since they generally indicate a marked involvement of the peritoneum.

In many cases there is temporary difficulty in micturition, so that a catheter must be employed. Frequency of urination occurs in those cases in which the abscess points toward the bladder. There is usually a distinct increase in the amount of indican in the urine.

A question of the greatest clinical importance is whether the perityphlitis will lead to the formation of a large abscess or not. We have already said that in a pathological sense almost all cases of perityphlitis are suppurative, but the formation of a large collection of pus is of practical importance.

The temperature conditions are most significant in determining the question of abscess formation. If the fever lasts more than a week or a week and a half, suppuration is highly probable. If there is a temperature of 104° to 105° F. (40° to 40.5° C.) we may suspect suppuration, even at an early period, particularly if the rate of the pulse corresponds to the height of the fever. Rigors are not frequent at all, but, if they do occur, they indicate abscess formation. The local signs, on the other hand, are not very valuable in the diagnosis of an abscess. Early in the attack it is scarcely ever possible to detect fluctuation, for the abscess is deeply situated and surrounded by infiltrated tissue.

A definite conclusion can be reached in many cases by exploratory puncture,

but the latter is generally only rarely used. On the other hand, it is very important to determine the number of leucocytes in the blood (Curschmann). With increasing leucocytosis (16,000 to 20,000 leucocytes or more to the cubic centimeter) suppuration is most probable. However, this sign can, of course, only be utilized in conjunction with all the other symptoms.

The further course of perityphlitis is favorable in the overwhelming majority of cases. After a few days, or a week or so, if the treatment is appropriate, the symptoms abate. The fever disappears, as already mentioned, either gradually or, not infrequently, quite rapidly, almost like a crisis. The inflammatory tumor shrinks, the pain abates, the bowels become regular, and the patient gradually regains complete health. On the other hand, often there are manifold variations in the disease, by which its duration may be prolonged to two and three weeks and over. But, even then, complete restitution occurs frequently enough without further intervention.

With regard to the pathological changes in all these cases that recover, of course we have no accurate knowledge. Sometimes it is probable that there never is a well-marked abscess formation, but only an inflammatory infiltration of the tissue, which subsides again. It is probable that, in many cases, there is a small abscess which discharges spontaneously into the intestine, usually into the cæcum, but exceptionally into other portions of the intestine. One thing which seems to us to suggest such a spontaneous discharge of an abscess is the not infrequent occurrence of a crisis, with rapid fall of temperature and simultaneous cessation of all the other symptoms. Still, we scarcely ever detect pus in the stools, for the pus corpuscles are changed during their passage from the colon to the rectum. Perhaps in some cases small localized abscesses are also reabsorbed.

On the other hand, an unfavorable termination is certainly not infrequent, and this fact explains the widely spread popular opinion that appendicitis is a dangerous and treacherous disease. The most dangerous cases are those in which a diffuse septic peritonitis develops at the onset in consequence of an extensive gangrenous perforation of the appendix before any adhesions have formed. These are the cases which impress one as being very serious from the start, and which end fatally in a few days. The primary focus of the peritonitis may readily be entirely overlooked. Occasionally the disease may commence as an apparently harmless simple appendicitis, and on the second or third day the mild clinical picture may suddenly change to the grave one of general peritonitis with an almost always rapidly fatal termination. In a third class of cases, the disease is at first of moderate severity, but after a few days shows a marked improvement, so that one is led to expect a spontaneous recovery. Then, suddenly, a turn for the worse ensues, either rapidly or more slowly (renewed elevation of temperature, vomiting, peritoneal irritation, etc.), and, unless there is prompt surgical intervention, the danger of generalized peritonitis from a rupture of the abscess into the peritoneal cavity is very great. In other cases, nowadays very rare, the abscess is left to itself and burrows a way for itself. Either it finally breaks through the abdominal wall, or it may extend downward into the iliac fossa or the groin [or upward toward the liver], or perforate into the rectum, bladder, or other parts. It has also happened that the suppuration has involved the ileocæcal vein, with consequent pylephlebitis and the formation of

abscesses in the liver. In most of these chronic cases the symptoms are those of a tedious septicopyæmia such as is nowadays, thanks to the progress of surgery, very rarely seen. As the inflammation originates from perforation of the intestine, the pus is apt to be extremely offensive whenever there is any extensive abscess formation.

Finally, we must add that, in isolated cases of acute appendicitis, symptoms of intestinal obstruction (*vide infra*) may develop, such as tympanites, frequent eructations, or even fecal vomiting. Complications in other organs are exceptional.

Diagnosis.—In general, there is no difficulty in the diagnosis of perityphlitis. Nevertheless, the differential diagnosis between appendicitis and an acute gallstone or renal colic will occasionally have to be considered. In women the differential diagnosis between appendicitis and acute inflammations in the neighborhood of the uterus (parametritis, etc.) is not always simple. Finally, we ourselves for several days had regarded an attack of appendicitis without marked local symptoms in a child as a case of typhoid, until the diagnosis became clear from the local conditions.

The principal difficulties in the diagnosis consist in estimating the particular anatomical conditions, the formation of an abscess, its position, etc. A thorough examination in every way (*per anum* also) is indispensable. In so far as this relates to the extremely important question of surgical interference, we shall revert to the matter when considering treatment. In those rare cases of perityphlitis which have a chronic course, the disease may be confounded with new growths, particularly carcinoma originating in the cæcum or the appendix. Mistakes in diagnosis have also been occasioned by tumors of the right kidney and the right ovary, and by psoas abscess due to Pott's disease.

Tuberculosis, and in rare cases, actinomycosis, may also produce clinical pictures similar to those of appendicitis. In this connection it may be mentioned that in rare cases the lumen of the appendix may become occluded, giving rise to so-called dropsy of the vermiform appendix. This may occasion a tumor which can be felt in the ileocæcal region.

Prognosis.—In every case of perityphlitis the prognosis should be guarded, since the course of the disease cannot be foreseen. Nevertheless, in by far the greater number of cases, the termination of the acute perityphlitic attack is a favorable one, and the lighter cases, with circumscribed inflammation, are the rule. If we take all the cases of acute inflammation of the ileocæcal region, we shall find that some eighty or ninety per cent get well without operation, but still, it should be emphatically stated that perityphlitis is a dangerous disease, and every year it brings no small number of individuals, both children and adults, to sudden death. The main danger lies in the rapid onset of general peritonitis. Even after the patient has survived the attack of acute perityphlitis he may be troubled in various ways. For instance, the resultant adhesions and other chronic inflammatory changes may impede the action of the bowels for a long while, may cause localized pains, etc. In such cases one must always be prepared for a renewed onset of severe, acute symptoms, which may occur quite suddenly. Chronic appendicitis is like a quiescent volcano, from which, at any time with no assignable or some comparatively insignificant cause, such as constipation, slight trauma, or perhaps a second infection, a fresh outbreak

may be expected. Sometimes, as has already been mentioned, these chronic changes make themselves felt through constant mild subjective symptoms in the ileocæcal region. In other cases, the subjective symptoms cease entirely, and, notwithstanding, after a shorter or longer interval, there is another acute attack. In this way some people suffer four or five times, and even oftener, from acute perityphlitis.

Treatment.—As soon as the diagnosis of appendicitis or perityphlitis has been made or is even considered, the main indications are immediate complete rest in bed and the strictest diet (a little cold milk or soup, interdiction of all solid food). A not too heavy ice bag is placed over the ileocæcal region. If this is not well borne, a Priessnitz or a twenty-per-cent alcohol compress is prescribed. As a rule, a laxative is not necessary. Recently, however, there is an increasing tendency again to permit the careful use of castor oil (formerly so much in vogue), when constipation is present at the onset, provided there are no symptoms of peritoneal irritation. At present, opium is prescribed in most cases from the onset. If there are marked pains in the ileocæcal region, the action of the opium (tr. opii simplex, 5 to 10 drops, and more according to the age of the patient) is generally very favorable. A countercurrent against prescribing opium has arisen only from the fact that opium relieves the pain, and in this way conceals the danger of the onset of peritonitis, so that the proper moment for surgical intervention can easily be overlooked. In our opinion, this is not a sufficient reason to exclude opium entirely in the treatment of perityphlitis. But, on the other hand, opium should not be needlessly administered (i. e., without marked pains) in too large doses.

With these remedies—viz., rest, diet, ice, and opium—everything has been done to favor spontaneous cure in favorable cases. As has been already said, many cases recover under this treatment. There is no necessity at first to be anxious about opening the bowels. Usually there is no harm if the patient goes without a movement for five to eight days, providing that his condition is satisfactory as regards his general health, pulse, and temperature. It is indeed not unusual to have a natural operation of the bowels while rather large doses of opium are being administered, but if the abdomen becomes more and more distended and there is rather long-continued constipation, we need not hesitate to make a cautious use of glycerin suppositories or injections. For injection we usually choose oil or soapsuds, and let it run through an ordinary fountain syringe into the colon, while the patient is lying on his back. If necessary, the injections must be repeated, and of the beneficial effects of these oil or soap injections we have often been convinced.

We have already insisted that every patient with perityphlitis should be most carefully watched, however favorable the course of the disease seems to be. The physician must ask himself every minute whether further symptomatic treatment is permissible, or whether it may not be necessary to have the abscess opened by surgical interference.

Since the course of the disease cannot be told beforehand, and since the dangers of the operation are naturally smaller in proportion to the promptness of its performance, many eminent surgeons are of the opinion that in every case of perityphlitis the *corpus delicti* (the inflamed appendix) should be removed if possible on the first day, or, at the latest, on the second day of the disease, before it can do any further damage. The results of these early operations are

very good (casualties, it is true, do occur in isolated cases). Most of the cases would certainly also have recovered without operation. But in some cases the termination would have been unfavorable without operation, and most of those operated early are now safe from later recurrences, which is a great advantage. Nevertheless, the obligatory early operation will always remain more a theoretical demand than a procedure which can be practically executed. At any rate, numerous cases seek medical aid only on the third day or even later, and then the question whether we should operate at all and when, is frequently difficult to decide. The general answer to this question can only be that immediate operation is always indicated as soon as the presence of a perityphlitic abscess has been assumed, the absorption or spontaneous rupture of which into the intestines should not be waited for, because of the possibility of a rupture into the free peritoneal cavity with subsequent diffuse suppurative peritonitis, or of a general septic intoxication, or a protracted chronic suppuration. When this danger point is reached is often impossible to say with certainty; nevertheless, there are a large number of important indications. In general, we rely personally on the following rules: If the temperature last longer than five or six days without remission, or, particularly, if after a febrile attack during the first few days, a renewed elevation of temperature occur, then there is strong suspicion of an abscess formation. If the pulse rate (despite the low temperature!) rises, and it increases to 100 to 120 while the tension decreases, this also is no favorable sign. Continued eructations and recurrent vomiting are likewise unfavorable. Accurate observation of the local condition (tumor, increasing tenderness) is very important, but attention to the general condition (pallor and bad look, dry tongue, beginning facies abdominalis) is of equal importance. Finally, the leucocytosis should be considered (*vide supra*). An uninterrupted increase of the leucocytes to 18,000 to 20,000 to the cubic millimeter and over, always increases the suspicion of an existing suppuration. On the other hand, a low leucocyte count should not keep us from operating, if the condition is otherwise unfavorable.

It is impossible to give more detailed rules, of general application, with regard to the question of operating. The individual circumstances of each particular case and the personal experience of the physician must always be controlling factors. No physician can avoid unfortunate occurrences in perityphlitis, and the most experienced practitioner will never escape a certain feeling of uneasiness in any case of the disease.

In general, during the first few days of the disease (excepting cases of possible early operation), if the general condition of the patient is good, expectant treatment is certainly to be recommended; and even in the ranks of the surgeons the eagerness for operation has grown less in this disease, just as it has in many others. On the other hand, operation must not be delayed in those cases in which severe local symptoms or general septic intoxication occurs within the first few days; and in every case, as Sahli so aptly puts it, expectant treatment should always be an "armed truce." For this principle is certainly true: better to operate too early than too late.

If the attack has passed, the question of an interval operation (*"à froid"*) arises. Since in many cases adhesions of the appendix persist, or a fecal concretion, or even a small inspissated pus focus, remain in the appendix, a constant danger of recurrence (*vide supra*) is thereby occasioned. This danger is obvi-

ated once and for all by the removal of the appendix. The operation is to be particularly recommended, if several, even light, attacks of appendicitis or perityphlitis have occurred, or if any remaining slight discomforts (pains, tenderness on pressure, palpable appendix, irregular bowels, etc.) indicate persisting morbid changes in the appendix. There can hardly be any doubt that many accidents can be avoided by an interval operation in which, as a rule, there is almost no danger.

Moreover, for the after-treatment of perityphlitis, mild Carlsbad cures, mud packs, careful massage, etc., are still occasionally employed.

CHAPTER V

PERFORATING ULCER OF THE DUODENUM

There is a form of ulcer of the duodenum, especially of its upper, horizontal portion, which is precisely analogous to the round gastric ulcer in regard to ætiology, pathological anatomy, and, very largely, symptomatology. The ulcer is probably also due in most cases to the action of the acid gastric juice on the duodenal mucous membrane under conditions which have been detailed more fully in the ætiology of gastric ulcer. We must mention here the noteworthy fact that, after extensive burns of the external skin, ulceration of the duodenum, rarely of the stomach also, has been repeatedly observed. This is probably due to the thrombosis of a duodenal vessel, occasioned by the setting free of fibrin ferment.

Ulcer of the duodenum is much rarer than the round gastric ulcer, and, in distinction from the latter, it has been found decidedly oftener in men than in women.

Many cases of ulcer of the duodenum run their course entirely without symptoms, or they cause no symptoms until sudden hemorrhage appears (from erosion of the pancreatico-duodenalis, gastroduodenalis, etc.) with hematemesis and bloody stools (quite often bloody stools alone without hematemesis), or until there are suddenly signs of peritonitis from perforation. In many cases a type of disease exists for a long time whose symptoms, as we have said, are so like the clinical symptoms of gastric ulcer that we can only rarely distinguish the two forms with certainty during life. We notice continuous or neuralgic pain, which, in ulcer of the duodenum, has its chief seat in the right hypochondrium. Severe gastric symptoms, especially vomiting, are not as common as in gastric ulcer. Occult hemorrhages, demonstrated by the different chemical blood tests of the stools, may materially support the suspicion of an ulcer. Finally, it is to be noted that recently a slight glycosura has repeatedly been observed in duodenal ulcer (in contradistinction to ulcer of the stomach). The general health and general nutrition may remain quite undisturbed for a long time.

Ulcer of the duodenum ends by cicatrization and recovery, or by cicatrization and the formation of stenosis, and obstructed evacuation, with secondary dilatation of the upper portion of the duodenum and of the stomach. In regard to the different adhesions and the perforations of the ulcer into neighboring organs, we may refer to what has been said of gastric ulcer.

The treatment must be governed by the same principles which were laid down in the treatment of gastric ulcer, especially as the diagnosis is usually doubtful.

CHAPTER VI

TUBERCULOSIS OF THE INTESTINES

Tuberculosis of the intestines is in most cases a secondary disease, and is one symptom of a more extensive general tuberculosis. It develops most frequently in the course of chronic pulmonary tuberculosis, and depends here, as we have seen (compare page 307), probably upon an infection of the intestines from the tuberculous sputum that has been swallowed.

Intestinal tuberculosis, however, may also be a primary disease, and the source of further extension of tuberculosis over the body. "Tuberculosis of the abdominal organs," which usually starts from the intestines, has a clinical significance, especially in children. It is not improbable that in such cases the first infection of the intestine comes from without, and that the tuberculous poison is taken into the body with the food. Here we must suspect especially the milk from cows with pearly distemper—that is, with tuberculous disease.

The anatomical changes in intestinal tuberculosis are precisely analogous to the tuberculous changes in other mucous membranes. The tubercular new growth has its origin usually in the lymph apparatus of the intestine, in the solitary follicles, and in Peyer's patches. The first miliary tubercles form beneath the epithelium, and soon fuse with one another into a diffuse infiltration. In its further course the infiltration on one side extends deeper into the surrounding tissue, so that it attacks the submucous and muscular coats and even extends to the serous coat, and on the other side, by the destruction of the new growth which begins at the surface and constantly spreads, tubercular ulcers are formed. We can often make out with the naked eye single miliary tubercles or groups of them at the base or in the infiltrated edges of the ulcer. This is especially plain in deep-seated ulcers on the corresponding portion of the serous coat. The form of the large tubercular ulcers is often irregular. In many cases the long diameter of the ulcer is parallel to the circumference of the intestine, so that the girdle-like ulcers, which are especially characteristic of tuberculosis, are formed.

Tubercular ulcers are situated both in the large and in the small intestines. They are usually most marked in the vicinity of the ileocæcal valve. Isolated tubercular lesions, which may impress one absolutely like an ileocæcal tumor, are not infrequent in this locality. The thickened, matted coils of intestine, in conjunction with the swollen lymphatic glands and other tissues, occasionally form large irregular tumors, which may give rise to confusion with chronic perityphlitis.

Tubercular ulcers in the stomach are extremely rare. Besides the intestinal tuberculosis there is very often tuberculosis of the mesenteric lymph-glands, and also frequently tuberculosis of the peritoneum.

The symptoms of intestinal tuberculosis are usually quite subordinate to the symptoms caused by other coexisting tuberculous affections. There may often

be quite extensive tubercular ulcers without any marked symptoms, but, as a rule, the onset of diarrhea turns the attention to the intestinal complication (see the chapter on Pulmonary Tuberculosis).

Marked admixture of blood with the stools occurs only rarely in tubercular ulcers of the intestine. In tuberculosis of the large intestine, blood and pus are occasionally found in small amounts in the stools.

Tubercle bacilli have been repeatedly demonstrated in the fecal discharges, but it is not very easy to find them, and, moreover, their diagnostic significance is impaired by the fact that they may be referable to sputum which has been swallowed.

Pains are often entirely absent. In other cases there is considerable abdominal pain as well as much tenderness on pressure. Marked tenderness on pressure indicates an involvement of the peritoneum. Not infrequently, a persistent moderate meteorism is present. To a certain extent, circumscribed ileocæcal tuberculosis furnishes a very characteristic clinical picture. It generally produces the symptoms of a chronic intestinal obstruction (colic, constipation, visible intestinal peristalsis), and is generally associated with a distinct circumscribed tumor in the ileocæcal region. The course is generally very protracted, lasting possibly for years. Secondary suppuration is not infrequent.

Primary tuberculosis of the abdominal organs sometimes presents quite a characteristic type of disease, especially in children. This was termed by the older physicians *tabes mesenterica*. The chief feature of this type of disease consists in a progressive general emaciation and anæmia, which are usually associated with a persistent hectic fever, which obstinately resists all the remedies employed. The abdomen is usually swollen by meteorism, but it is sometimes flat and sunken. In some cases we can feel the swollen mesenteric lymph-glands through the abdominal wall during life. The involvement of the mesenteric lymph-glands obstructs the lymph-channels which serve for the absorption of fat, a circumstance which contributes to the excessive emaciation frequently seen. The liver may be enlarged and its lower border can often be felt. The bowels are irregular, and there is usually a moderate diarrhea, persisting in spite of all remedies. The invariably fatal termination is due to an increase of the general marasmus, or to a final acute tuberculous affection, such as miliary tuberculosis or tuberculous meningitis. The autopsy shows tuberculosis of the intestines, peritoneum, lymph-glands, liver, etc., to a greater or less extent. The lungs may be quite free from tuberculosis. We will return to this affection in the description of tuberculosis of the peritoneum.

The treatment of intestinal tuberculosis can, as a rule, be only symptomatic. Besides the general dietetic treatment which, in addition to sparing the intestines, seeks to keep up the patient's strength as far as possible, medical interference is demanded by the abdominal pain and diarrhea. The chief remedy is opium, either alone or combined very frequently with tannin, tannigen, tannalbin, acetate of lead, subnitrate of bismuth, bismutose, coto bark (gr. vijss. [gm. 0.5]), etc. Sometimes a decoction of logwood is beneficial. Talc also has lately been recommended; about 6 or 7 ounces (gm. 200) are given daily, suspended in milk. We have seen some good results from it. The most serviceable local applications are poultices and alcoholic and Priessnitz compresses. In ileocæcal tuberculosis (*vide supra*), surgical intervention (extirpation, enteroanastomosis, etc.) has frequently given good results.

In other respects the treatment coincides with the general treatment of tuberculosis (*vide supra*).

CHAPTER VII

SYPHILIS OF THE RECTUM

In not very rare cases we see in the rectum, especially in its lower portions, extensive syphilitic ulcerations, which produce a severe and practically important type of disease. The more intimate relation between syphilis of the rectum and the general syphilitic process is not perfectly clear. According to quite a widespread opinion, the infection of the rectum comes from the secretion trickling down from the ulcers of the genitals. The facts seem to support this view, since syphilis of the rectum is seen much more frequently in women than in men. Some authors have even asserted that all the so-called "syphilitic" ulcers in the rectum have no connection at all with genuine syphilis, but are chancroids. It is, in fact, striking, even if it by no means proves such a hypothesis, that, at the autopsy of persons who have died of "syphilis of the rectum," we rarely find definite syphilitic changes in other internal organs—a fact which we also can confirm.

The most characteristic mark of syphilitic ulcers in the rectum is their tendency to form cicatrices and stenoses. This result of the ulcer is also important in its clinical relations, since the chief symptoms of the disease usually begin with the development of the stenosis. The seat of the stenosis is usually so low down that we can conveniently reach it with the finger, on a digital examination of the rectum during the patient's life. The rectum narrows like a funnel upward, and we can feel the quite sharp edge of the ring-like cicatrix with the point of the finger. This funnel-shaped stenosis of the rectum is so characteristic of syphilis of that organ that, in almost all cases, we can make the diagnosis with perfect certainty from this alone.

The rectum and the descending colon are usually dilated above the stenosis, and here extensive, irregular ulcerations, with undermined edges, are usually found in the mucous membrane. These are partly of a specific nature, and partly diphtheritic ulcers caused by the pressure of the accumulated fecal masses.

The symptoms of syphilis of the rectum usually develop quite gradually. At first the bowels are irregular, and there are disturbances of defecation which stubbornly resist the ordinary remedies employed. There are sometimes, in the first stage of the disease, frequent and severe hemorrhages with the dejections, as we have seen, and for a long time these may falsely be considered to be "bleeding from hemorrhoids." The symptoms become more marked as cicatrization of the ulcer increases and as stenosis of the rectum develops. There is usually a decided catarrh of the rectum, so that the thin stools contain a large admixture of mucus and pus. The patient's condition is extremely distressing, from the pains with the frequent but always scanty dejections, and from the severe tenesmus. Nodular thickenings and prolapse of the mucous membrane, and sometimes true hemorrhoids, form about the anus. The patient's strength constantly diminishes from the pain and the continual diarrhea. He finally

becomes emaciated, looks very pale and wretched, and has fever toward night. Death ensues from increasing general weakness, or rarely from a terminal peritonitis due to perforation, after the whole disease has lasted one and a half to two and a half years.

This unfavorable termination unfortunately seems to be the rule in all the cases described; hence the prognosis is to be regarded as very serious in all cases of syphilis of the rectum. Improvement worthy of mention, or even perhaps recovery, is possible only when the disease is recognized at the outset and properly treated.

At the outset of the disease the treatment, of course, must consist chiefly of an energetic general attack upon the syphilis by mercurial inunctions and iodid of potassium; but, when once the characteristic funnel-shaped stenosis of the rectum has formed, we cannot expect much from antisyphilitic medication, since this cannot exert any influence on the cicatrices and their results. Improvement is now to be obtained only by mechanical dilatation of the stenosis by bougies, or, if this is not enough, by a surgical operation.

Treatment with bougies, in order to produce results, must be carried out very carefully for a long time. The operative treatment consists in incision and dilatation of the stenosis, or in a complete resection of the diseased rectum; in certain cases, also, in the formation of an artificial anus. When only symptomatic treatment is possible, it consists of local irrigations, similar to those employed in catarrhal colitis, and in the careful administration of mild laxatives (rhubarb, compound licorice powder, castor oil, etc.).

CHAPTER VIII

CANCER OF THE INTESTINES

THE development of cancer is far more rare in the intestine than in the stomach. Nevertheless, cancer of the intestines is a disease of great practical importance, since a cure is possible only in case of a correct early diagnosis. The rectum is the most frequently affected of all portions of the intestines. Next in frequency is carcinoma of the colon, particularly the cæcum, the sigmoid flexure, and the hepatic and splenic flexures. Carcinoma is very much less common in the small intestine, though it occurs in all parts of it. In the duodenum it particularly affects the neighborhood of the common duct.

Most cancers of the intestine appear in the form of ringlike swellings that take in the whole circumference of the intestine. More rarely we find a more diffuse papillary proliferation, extending over a larger surface of the intestine. There is often quite an extensive destruction of the new growth on the surface of the cancer, from which deep ulcerations arise. Often metastases are found in the lymph-glands, liver, and other organs. Above a cancerous obstruction the intestine is usually dilated, with thickening of its muscular coat, and inflammation or ulceration of its mucous membrane due to the impacted fecal matter. In its histological structure, cancer of the intestine is to be regarded as invariably a cylindrical-celled carcinoma, which sometimes shows a plainly glandular structure—adeno-carcinoma—and sometimes that of the other forms of cancer—scirrhous, medullary, or colloid.

Cancer of the intestines, like all cancers, occurs chiefly, if not invariably, in advanced life.

Symptomatology.—The clinical symptoms of cancer of the intestines are only in a part of the cases so pronounced that we can make a positive diagnosis of the disease. The symptoms vary with the location of the growth.

Cancer of the rectum begins usually with distress at stool (tenesmus) and pain in the rectum, which at first comes only with defecation, but later becomes almost continuous. The disturbances of defecation consist at first apparently only in simple constipation. Often there is frequent desire to defecate, the movements consisting always of small tapelike or nodular masses, which are evacuated with great difficulty. Gradually a painful tenesmus sets in. The pain often shoots into the neighboring parts—the sacrum, the thighs, the genitals, etc. The local symptoms gradually increase, the stools often contain some mucus and blood, and diarrhea alternates with obstinate constipation. The patient also becomes emaciated, and constantly grows weaker and more miserable. Finally, we often find a complete paralysis of the sphincter ani, so that a mucous, bloody fluid constantly oozes from the half-open anus. An absolute diagnosis can usually be made with ease by digital examination of the rectum. This examination should never be omitted in any suspicious case, or otherwise it is only too easy to make a mistake. Many a fatal case might perhaps have been saved by a timely diagnosis. External piles are common in cancer of the rectum, and their presence should by no means prevent the physician from an internal examination. If there is cancer of the rectum, the finger upon being introduced detects an irregular, firm growth, and we can usually make out with approximate accuracy its extent and mobility and its invasion of neighboring organs, such as the vagina and bladder. We differentiate cancers of the rectum into those situated far up (generally markedly exuberant), and those located low down (generally annular). Examination with the rectal speculum sometimes makes the diagnosis more accurate, but is usually unnecessary. In some cases the destruction of the new growth may cause perforation into the adjoining organs mentioned, and we can easily understand the results of this, such as cystitis, purulent discharges from the vagina, etc. We may also have peritonitis from perforation. Secondary cancer appears with especial frequency in the liver, also in the peritoneum and elsewhere.

Carcinoma of the colon causes, as a rule, only very indefinite symptoms, which for a long time are hard to interpret. These symptoms consist chiefly of distress at stool, obstinate constipation, dull pains in the abdomen, and the signs of slowly increasing general weakness and emaciation. With the growth of the neoplasm there are usually attacks of colic, with the gradual or perhaps rapid development of all the symptoms of obstruction of the bowels (*vide infra*, Chapter XI). The pains are rarely sharply localized; but sometimes, however, the principal location of the pain, and particularly the localized tenderness, point approximately to the seat of the neoplasm. Not infrequently the pain is principally referred to the umbilical region. Vomiting occurs fairly often in cancer of the colon. At first the vomitus consists of stomach contents, mucus, bile, later (with increasing intestinal obstruction) it may assume a fecal character.

In many cases the shape of the feces is peculiar. They are ribbon-shaped, or consist of queer flattened nodules which have a certain resemblance to sheep's

dung (scybala). Not infrequently we find in the dejections an admixture of mucus, pus, and, what is still more important, blood; if blood is persistently present in the movements (to be determined by the guaiac-turpentine test, *vide supra*), or if there are bloody or mucopurulent masses in the discharges, cancer is suggested. Exceptionally there may be found in the stools small detached portions of the new growth. Not very rarely in cancer of the intestines the local symptoms for a long time remain in the background; only an increasing general weakness, or a striking anæmia, arouse suspicion of a more serious affection, the true nature of which remains unrecognized for a long time, since the most careful examination of the abdomen frequently at first yields no result. However, on careful palpation it is sometimes possible to feel the new growth through the abdominal walls as a distinct tumor. One characteristic of the tumor occasioned by intestinal carcinoma is that it may be very movable, and change its position because of its own weight or of the collections of fecal matter. As a rule, the growth does not move with respiration, except possibly in carcinoma of the transverse colon. Most cancers of the colon are more distinctly felt with an empty gut than with a full one. Insufflation of air through the rectum may facilitate the exact localization of the seat of the neoplasm in isolated cases. In doubtful cases an examination under narcosis is indicated.

It is easy to confound colon carcinoma with carcinoma of the stomach, the mesentery, the mesenteric lymph-glands, kidney, etc. It is very important to know that in case of intestinal carcinoma the tumor which is felt may be not the new growth at all, but the hardened fecal masses collected above it. Such fecal accumulations may cause all sorts of errors as to the situation and dimensions of the new growth. In such cases a correct conclusion may not be obtained until after continued observation and the use of laxatives and enemata. It is also true that ordinary fecal masses may be mistaken for cancer of the intestine when there is no intestinal disease of any sort. If the neoplasm increases in extent, the symptoms of intestinal stenosis become gradually more and more prominent (colic, vomiting, visible rigidity, distention, and peristalsis of the intestinal coils situated above the obstruction). The symptoms of stenosis may be relieved from time to time, owing to ulceration of the cancer. In exceptional cases there may be perforation of cancer of the colon into the stomach, with the formation of a gastrocolic fistula; of perforation of cancer of the sigmoid flexure into the bladder or into the peritoneum, with a secondary peritonitis, which may be localized or diffuse. Carcinoma of the cæcum is often indistinguishable for a long while from tumors due to chronic typhlitis or perityphlitis. Cancers of the appendix may also be confused with a circumscribed chronic tuberculosis of the ileocæcal region. Cancer is suggested, above all, by the age of the patient, the progressive aggravation of the illness, and sometimes also by swelling of the inguinal lymph-glands. In the surgical wards in Leipsic we observed a case of cancer originating in the vermiform appendix, which penetrated through the skin.

Cancer of the small intestine is very rare. It is distinguished by the very great mobility of the tumor, if one is found. The symptoms are those of gradually increasing stenosis (*vide infra*). Cancer of the duodenum is similar in many respects to cancer of the stomach, particularly of the pylorus. It leads to stenosis of the duodenum, and consequently to dilatation of that portion of

the duodenum which is above the new growth, and of the stomach, and so occasions the well-known symptoms of gastrectasia. Cancer which is seated in the neighborhood of the common duct usually causes excessive and persistent jaundice.

Prognosis.—The course of all cases of intestinal cancer, provided operative removal of the tumor is impossible, is absolutely unfavorable. The disease may last for a considerable time, about two or three years. On the other hand, the duration of the special symptoms may be comparatively brief—that is, a few months or weeks; undoubtedly because the condition existed long before it caused symptoms. Most of the cases run their course under the clinical picture of a more or less slowly developing intestinal stenosis, which may lead to almost complete obstruction. The final fatal result in intestinal carcinoma takes the form either of increasing general weakness or of perforation with fatal suppurative peritonitis. Death may also be occasioned by extensive and foul suppuration in the surrounding connective tissue, or by phlebitis and pyæmia.

Treatment.—The only possibility of successful treatment in intestinal carcinoma is by surgical methods. The results of operation are very favorable in cases of cancer of the rectum if the disease is not situated too high, and has not extended too far. Cancer of the other parts of the intestine has a less promising outlook. Still, a large series of permanent cures have already been obtained by resection of intestinal cancers. In isolated cases, with stenotic symptoms, good palliative results have also been obtained by means of the so-called entero-anastomosis. If surgical interference is impracticable, treatment must be merely symptomatic (suitable diet, mild laxatives, narcotics, hot compresses, etc.), with a view to alleviating suffering.

We will append a few remarks about *sarcoma of the intestine.* This is a very rare disease, which originates from the submucous layer, or else, in the form of lympho-sarcoma, from the lymph-follicles of the intestine, particularly of the small intestine. Both varieties exhibit a tendency to extend cylindrically over considerable areas along an intestinal coil. They usually do not lead to stenosis, and they may even cause dilatation of the affected portion of the intestine. The tumors which are formed may attain to very considerable size. The most prominent symptoms are general weakness and anæmia, associated with slight fever. These constitutional symptoms are more prominent than the local ones. The course is usually rapid, and ends, after six to twelve months, in death.

CHAPTER IX

HEMORRHOIDS

By the name "hemorrhoids" we mean diffuse or varicose dilatations of the hemorrhoidal veins, especially of the venous plexuses at the lower end of the rectum. Hemorrhoids are single large varices, which usually rise from the submucous layer, and push the mucous membrane out before them. If they are situated outside of the sphincter ani we speak of external hemor-

rhoids, in distinction from internal hemorrhoids, which lie above the sphincter. The size of the nodules varies with the fullness of the dilated veins; but hemorrhoids, as a rule, do not consist exclusively of dilated vessels, for we often find, at the same time, considerable thickening of the surrounding connective tissue, so that the whole mucous membrane has a swollen appearance, with a polypoid proliferation in parts. The hemorrhoids usually present themselves as bluish tumors, from the size of a pea to that of a walnut, which surround the anus like a garland. Most of them have a broad base, while others are apparently pedunculated.

The chief cause of hemorrhoids is frequently repeated stasis in the veins affected. The position of the hemorrhoidal veins is such that their contents are constantly pressing downward by the action of gravity, and the development of varices is further promoted by their complete lack of valves. Special local conditions often contribute also to render the escape of blood difficult; thus hemorrhoids are particularly frequent during pregnancy, also in persons with habitual constipation, and consequently in individuals who follow sedentary pursuits. Local disease of the mucous membrane of the rectum (catarrh, neoplasms, etc.) also gives rise in some cases to the formation of hemorrhoids. Hemorrhoids are sometimes found as a result of portal congestion (in hepatic cirrhosis and similar conditions), and finally in general circulatory disturbances due either to pulmonary or cardiac disease; but these cases are not so frequent as was formerly supposed. Quite often, however, we can discover no sufficient cause for the development of the disease, and we are then forced to the hypothesis of a local disease of the affected venous plexus, which is probably often connected with an individual, and apparently sometimes hereditary, predisposition. We most frequently see hemorrhoids in men in middle life.

Hemorrhoids sometimes cause only slight symptoms, or none at all, but in other cases they are a tedious, burdensome, and even distressing evil. The chief symptom is pain, which is felt as a constant burning at the anus, but which increases to great severity at each dejection. There is much pain when the hemorrhoids and the surrounding tissue gradually get into an inflamed condition. In the skin about the anus, erythema, excoriations, and sometimes small but very painful fissures are formed. The mucous membrane at the lower end of the rectum is often found in a catarrhal state, which gives rise to the presence of pus and mucus in the dejections—"mucous hemorrhoids." The worst symptoms are caused when by pressing and straining at stool an internal pile is forced outward and contricted by the sphincter. If the constriction is not speedily relieved the extruded varix becomes much swollen and inflamed. This change is favored by a cramp-like contraction of the sphincter. Under such circumstances there may result a purulent phlebitis associated with very great pain, high fever, and severe constitutional disturbance. Sometimes we find a thrombus, thrombophlebitis, or suppurative periproctitis, even if there is no previous constriction.

Hemorrhoidal bleeding is a frequent and familiar symptom. It arises from small fissures of the dilated and thinned venous walls, and is frequently produced by defecation. Sometimes it occurs at apparently regular intervals, but more frequently it follows absolutely no rule. The hemorrhage is seldom very large, so that the loss of blood is in itself very rarely dangerous. Still,

the rectal hemorrhage may be repeated so often as to cause severe and persistent anæmia. The diminution in size of the varices after the hemorrhages explains why the hemorrhoidal symptoms are apt to be less marked as long as there are hemorrhages than when there are none. Hence the old term of the "golden vein" for hemorrhoidal bleeding.

Besides the local symptoms mentioned in the anus, there are sometimes other symptoms which are due to an implication of the neighboring venous plexuses, the vesical, prostatic, and sacral plexuses. There is often pain in the sacral region, difficulty in micturition, and in women vaginal catarrh, anomalies of menstruation, etc. Since the symptoms of some coexisting morbid conditions, such as abnormal corpulency, chronic gastro-intestinal catarrh, general neurasthenia, etc., may be added to the general picture, we can comprehend why medical superstition has found in hemorrhoids an excuse for the strangest ideas, like that of "aberrant hemorrhoids"! The scientific physician should be careful not to refer such manifold symptoms to hemorrhoids without due consideration, for otherwise important diseases may be readily overlooked.

Treatment.—The treatment of chronic piles is, in the first place, mainly dietetic. The diet to be prescribed should be determined by the constitution of the patient. It is usually advisable to limit the ingestion of meat, and to recommend in its stead a larger proportion of vegetables, in a broad sense, including fruit, such vegetables as grow below the surface of the ground, simple puddings, and buttered bread. It is usually advantageous to prescribe a sufficient amount of physical exercise. In addition to regular walks, gymnastics, home exercises, rowing, etc., are to be considered, while riding and bicycling, on the other hand, are not well borne by many on account of the local irritation they occasion. The principal requisite is to insure regular and easy evacuations of the bowels. In addition to the general dietetic measures just mentioned, cathartics are often required. The bitter waters (Friedrichshaller, Ofner, etc.), sulphur (the principal ingredient of most "pile powders,"—e. g., sulphur depurati, tartari depurati), āā ℥ss. (gm. 15), elaeos, menthæ,[1] ℥ijss. (gm. 10), one half to one teaspoonful s.o.s., and compound licorice powder, are preferred as particularly reliable. Enemata are to be employed only with care on account of the local irritation, but small cold injections, or oil injections, may occasionally be of use. A favorable combination of all necessary dietetic requirements is often to be had at some health resort. Of these, Marienbad, Carlsbad, Tarasp, Kissingen, Homburg, etc., are to be principally recommended.

The local treatment consists chiefly in protecting the piles from irritation. The parts are best cleaned after every movement of the bowels with cotton or soft linen, and the region of the anus is to be touched with oil, vaselin, or borated vaselin. Regular washings of the anal region with solutions of boric or tannic acid are also to be recommended. Hip baths, the temperature of which may be regulated according to the subjective experience of the patients, have also a favorable action. If there are slight inflammatory symptoms we apply compresses wet with a solution of acetate of lead, or ice water, or try suppositories containing morphin or cocain. Certain ointments (e. g.,

[1] Powdered sugar, ℥j; volatile oil, gtt. ij. M.

chrysarobin, extr. belladonnæ, āā gr. xv. [gm. 1], iodoform, gr. vijss. [gm. 0.5], vaselin, 3v [gm. 20]), are also highly raised. We have repeatedly seen very good results from the so-called anusol suppositories (anusol-iodin-resorcin-sulphonate of bismuth), one or two of which are introduced into the rectum daily. The so-called noridal suppositories have a similar action. Anæsthesin or cocain ointments are recommended for severe pains. Constricted piles we should endeavor as soon as possible cautiously to replace with the finger. The pain attending this operation is often so severe that chloroform narcosis, or at least the local application of cocain, is required. If there is suppurative phlebitis an incision is necessary. In some cases a radical operation for the piles may be performed at the same time.

The bleeding from piles is, as we have said, seldom severe enough to demand special treatment, such as the use of ice, chlorid of iron, or packing the rectum (cotton tampons with 0.5- to 1-per-1,000 adrenalin solution). Internal hemostatic remedies (particularly the fluid extract of hamamelis, a teaspoonful three times daily, and hydrastis canadensis, ergotin, etc.) are entirely unreliable in their action. If the hemorrhage is repeated and serious, or if the inflammation causes persistent and extreme discomfort, we should urgently recommend operative removal of the piles. Particulars as to the methods employed for this purpose will be found in the text-books on surgery.

CHAPTER X

HABITUAL CONSTIPATION

Persistent tendency to constipation is a frequent symptom in many different acute and chronic cases. We often see persistent sluggishness of the bowels in all sorts of conditions of weakness, in general anæmia, in diseases of the stomach, intestines, liver, lungs, heart, and nervous system, and it is caused by a great variety of circumstances. Often the weakness of the peristalsis of the intestine, and particularly of the colon, is merely one manifestation of a general debility; in other cases the activity of the intestine is subnormal, because of the insufficient and unstimulating diet, or because of the lack of physical exercise. In a third class of cases there is disease of the intestinal wall itself, such as catarrhal conditions, circulatory disturbance, atrophy of the muscular coat, or constriction of the lumen of the intestine. And, finally, a fourth cause is disturbance of the innervation of the intestine, because of disease of the brain or spinal cord.

The above are examples of symptomatic constipation, but in this chapter we shall not discuss these, but rather cases of habitual constipation occurring as an apparently independent disease. This may appear alone or associated with other disturbances likewise purely functional. The causes of this frequent and important condition are by no means invariable, and we are still far from a clear understanding of the nature of all the disturbances that belong in this category. Sometimes habitual costiveness seems to be associated with a congenital functional weakness of the muscular coat, or perhaps of the nervous apparatus of the intestines. At any rate, it is often possible

to trace the symptoms back to the earliest childhood. In other cases it is apparently an improper mode of life which exercises a disturbing effect upon the intestinal activity; lack of bodily exercise often leads to disorders of the bowels, and, still more, irregularity in eating and an improper choice of food. In young girls, especially, false modesty may primarily lead to irregularity of the bowels.

Sometimes external mechanical influences promote constipation. In women unsuitable apparel, tight bands around the waist, tightly laced corsets, and the like certainly produce in many cases a permanent malposition of the intestinal organs ("enteroptosis," compare the chapter on Gastroptosis, page 565), and particularly of the large intestine. It is not improbable that these conditions are in part the cause of the chronic constipation which is so frequent in women and girls. In women frequent pregnancies may also exert a causative influence by inducing laxness of the abdominal walls and a tendency to enteroptosis.

Habitual constipation is associated still more frequently with general neurasthenia than with any of the conditions thus far enumerated (compare the chapter on Neurasthenia in Vol. II). The nature of this association is probably not the same in every case. Often neurasthenia and constipation seem to be coördinate symptoms; sometimes the existing constipation has an unfavorable effect upon the psychical condition of patients, rendering them feeble, fretful, out of sorts, and nervous. As a rule, however, neurasthenia is the primary disease, and then the irregularity of the bowels appears as a result of abnormal nervous influences or of secondary conditions (*vide infra*). Often the two states act in a vicious circle, each sustaining and aggravating the other. The most essential factor is often the hypochondriacal tendency of the patients. They ascribe an excessive importance to irregularity of the bowels, and regard every slight disturbance as of extreme gravity. Even when the movements are sufficient they cannot free themselves from a disordered apprehension that they are not really adequate. Such patients come at last to devote almost all their thoughts to their illness and make the most painstaking observations and investigations of their dejections, having energy for nothing else and no joy in life. They seek aid from all sorts of physicians and quacks, and yet they have no actual confidence in any one and do not follow the practitioner's directions with the requisite persistence. In many of these cases the constipation is only apparent or imaginary. The patient fears to eat any hearty food and takes little nourishment, and consequently the excreta are scanty. In other instances, however, the abnormal psychical conditions have a direct inhibitory influence upon the intestinal activity. The intestine gets out of the habit of regular action. Moreover, the unwise use of internal laxatives may entail actual disease of the intestine.

Consequently, if the physician wishes to form a reliable opinion with regard to the intestinal function in a case of chronic constipation from actual observation, the first requisite is that he should himself see the dejections of the patient for some time, while the diet is normal and all laxatives are omitted. This is much easier in a hospital or sanitarium than in ordinary private practice. When a patient is under observation in this way it should be noticed whether the abdomen becomes much distended, and whether hardened

fecal matter can be felt in the colon or the sigmoid flexure. Furthermore, the dejections should be examined as to their amount and character. They may seem nubby, hard, or as if they were "burned," or of abnormally small diameter, or flattened, or covered with mucus. If we find that the evacuations are distinctly abnormal we infer that there is actual disturbance of the intestinal functions; while, on the other hand, our examinations often show that the complaints are exaggerated or imaginary.

It is considered very important by many physicians to distinguish between an "atonic" and a "spastic" constipation. In the former case the obstipation is due to peristaltic weakness, in the latter to conditions of tonic-spastic contraction of the intestines. Dejections that are thin, pencil-like, or resembling sheep-dung, a retracted abdomen, palpable contracted intestinal coils, etc., are cited as the symptoms of spastic constipation. This distinction has a certain theoretical and even practical importance, but, in our opinion, cannot be strictly maintained.

Treatment.—The treatment of habitual constipation is a difficult and often a thankless task, and it demands patience and professional tact. It goes without saying that we must first of all look for the causal factors. If we succeed in improving the underlying disease—as, for instance, the chronic gastro-intestinal catarrh, the chronic affections of the heart or lungs, the anæmic conditions, or certain nervous troubles—a regulation of the bowels often follows of itself. In ordinary habitual constipation we must first attend to the patient's diet. Since most of these patients also suffer from symptoms of nervous dyspepsia, they are usually very careful in their diet, and take only a little, easily digestible, and chiefly liquid food. It is no wonder that no good dejections follow such food. Improvement can be obtained in such cases only by eating plenty of food which can mechanically stimulate the intestine. Hence we must try to bring the patient back to ordinary "household fare"—to take, besides plenty of meat, a sufficient amount of bread, vegetables, etc. It is a very good thing to recommend especially certain kinds of bread, such as Graham bread or rye bread, and also larger amounts of butter, besides fruits, prunes, grapes, apples, pears, dates, and figs, either raw or boiled, preserved fruits, and honey.

As a rule, the diet most suitable for each individual case is only determined by repeated trials. A coarse diet is not always the most suitable. In cases with susceptible stomachs, and those in which spastic conditions (*vide supra*) of the intestines must be thought of, results are occasionally more readily obtained by a lighter form of diet, in which, however, vegetables, stewed fruit, honey, butter, buttermilk, etc., must always be given the preference. It is often efficacious to have the patient drink a glass of cold water in the morning before breakfast. Of true laxatives we should be very sparing, for the patient readily becomes accustomed to them, and the dose has to be constantly increased. Among the milder laxatives the various waters which contain Epsom salts, such as Friedrichshall, usually one or two wineglassfuls are prescribed.

In general practice cathartic teas in the most varied combinations are much used with good results. We have personally used the following combination, e. g.: cort. frangulæ, folior. sennæ, herbæ millefolii, rhizoma gram. concis. āā ℥j ℥v (gm. 50), a teaspoonful to a cup of tea.

For more continued use we may also recommend tamarinds, rhubarb, podophyllin, aloes, jalap, cascara sagrada, and extract of frangula.

The purgen tablets (purgen = phenolphthalein) often produce good results, likewise California fig sirup (Califig.). In cases of so-called spastic constipation, good results are occasionally seen after the use of the preparations of belladonna (pills or powders with extr. belladonnæ,[1] gr. $\frac{1}{3}$ to ss. (gm. 0.02 to 0.03).

It is not possible in this connection to give a minute description of all these remedies. They are prescribed in the most varied combinations in the shape of pills and powders. It is often necessary to make repeated changes in the medicine and its dose before the most effectual prescription is hit upon, for there are many individual peculiarities in this regard. Generally the patients themselves decide what remedy answers their purpose best. There are patients with simple habitual constipation who for years take, every evening, some powdered rhubarb, or one or two laxative pills, with a good result and feel perfectly well. In most cases, however, the efficiency of the internal laxatives gradually fails, the patient is obliged to take more and more powerful remedies, or increasing doses, in order to get any effect. In such instances it is always best to abandon internal laxatives altogether, and in their stead to employ external physical agencies, or to stimulate the rectum by means of injections, irrigations, and the like.

The regular use of injections, if carried out in a proper manner, may be continued for a long time without harm. The practice may indeed be carried to an injurious excess, and occasion at last a catarrh of the rectal mucous membrane, because of the constant local irritation, or, at any rate, a needless dependence upon local stimulation. The simplest way of making an injection is by means of an irrigator. There are also various shapes of injection syringes used by many. For a fluid we choose simple warm water, or, what is often more efficient, cold water. We can strongly recommend the employment of soapsuds (a teaspoonful of powdered soap to a pint or two of water). Lately, injections of oil have been in great vogue. The patient is placed on his left side, and by the means of a fountain syringe, 8 to 16 ounces (gm. 250 to 500) of fresh poppy oil [or fresh cotton-seed oil] (olive oil is much dearer) is slowly introduced into the rectum. Small injections of glycerin, 1 to 2 drachms (gm. 5 to 10), are also effectual. Still more convenient and often satisfactory is the employment of glycerin suppositories, which are dissolved in the rectum by the heat of the body.

The external physical remedies employed in habitual constipation are massage, electricity, and hydrotherapy. Regular massage is often attended with good results. The patient is stroked with the flat of the hands with moderate or even with considerable pressure, beginning at the cæcal region, and proceeding along the colon to the sigmoid flexure. There may also be cautious kneading of the abdomen. A ball may be used for massage. This is very convenient, and can be carried out by the patient himself. An iron ball, weighing about five pounds and sewed up in flannel, is rolled for several minutes in a spiral course over the abdomen, its general direction being that of the hands of a watch. Where we have the necessary ap-

[1] [P. G. Ext. belladonnæ, U. S. P., is stronger.]

pliances, we may try vibratory massage, which often produces very good results.

The electrical treatment consists of faradization of the abdominal walls, or galvanization of the intestine, one electrode being placed upon the back. In the way of hydrotherapy, cold rubbing and sponging are employed, cool and "Scotch" (i. e., alternating warm and cold) douches, cold and warm compresses, etc. In addition to these methods medical gymnastics may be simultaneously practiced. The well-known books on indoor gymnastics (von Schreber and others) give detailed directions concerning exercises to strengthen the abdominal muscles and for the stimulation of intestinal peristalsis (raising the upper part of the body from the recumbent position, flexing the thighs on the abdomen, etc.).

In the treatment of habitual constipation associated with hypochondriasis, the first rule is to treat the patient's mental condition. We should not make merry over his trouble, nor should we rudely let him feel that we do not consider his complaints so important as he himself imagines. The patient does not deserve to be scoffed at, since his subjective symptoms are to him of the most urgent nature; but it is extremely important to divert his thoughts from his trouble. As in many other reflex processes, so in defecation, the voluntary attention abnormally directed to it has an inhibitory action. Hence we admonish the patient to think of his trouble as little as possible and to begin his regular activities again, and we try to convince him of the groundlessness of his fears. The cathartics, which most patients have already taken freely without the desired action, are usually of no advantage at all. On the contrary, it is almost always necessary to forbid the patient to use cathartics at all.

We should not only employ a suitable dietary (*vide supra*), but we should seek to stimulate the intestinal peristalsis by such external means as massage and electricity (*vide supra*). It is true that a considerable part of the success of these methods is due to their psychical effect upon the patient, but there is no doubt, also, of their direct invigorating influence upon the muscular coat of the intestine. We must not omit a proper general treatment: cold sponging, baths, a country residence, and sufficient physical exercise. Finally, it is often very useful to cause the patient to go to the closet at a fixed hour daily, even if there be no special desire for a stool, and to try to have a movement of the bowels. Thus, a sort of training and education of the bowels is achieved, even when the patient at first believes it impossible.

By these means only do we succeed in giving the patient renewed courage, and sometimes finally in attaining recovery even in severe and persistent cases. In all cases of habitual constipation we should, however, not only endeavor to hit upon the proper means for making the bowels act, but if it is in any wise possible we should also strive to arrange the patient's life so sensibly and suitably that he will have regular and satisfactory evacuations without any special means. To accomplish this, it is often requisite that we should persuade the patient for once to make the attempt to give up all laxatives. Such a course is naturally much more practicable in an institution than in a private house. The author has himself repeatedly treated patients with habitual constipation simply by regimen—i. e., by proper diet, cold sponging, and sufficient exercise or gymnastics, without any other remedies, regardless of the fact that

in such patients the stool would often be delayed for five, eight, or ten days, and yet, what is remarkable, without any special inconvenience. Then at last the bowels would move spontaneously, or with the aid of a small injection of cold water, and would gradually assume complete normal regularity.

CHAPTER XI

STRICTURE AND OBSTRUCTION OF THE INTESTINES

Ætiology and Pathological Anatomy.—The anatomical causes of stricture or obstruction of the intestines are as follows:

1. *Congenital Closure.*—Congenital closure of the intestines is found at the anus, *atresia ani,* and much less frequently in the colon or small intestines. The form first mentioned is the only one of clinical interest, since it may be relieved, at least in some cases, by operation. All the other forms of congenital closure of the intestines are incompatible with a long duration of life.

2. *Tumors and Cicatricial Strictures.*—Cancer of the intestine is the only tumor that has any clinical significance. We have already described its most important anatomical relations and the possibility of intestinal stenosis as a result of it.

We see cicatricial strictures most frequently after recovery from tubercular ulcers. They are located principally in the lower part of the small intestine or in the cæcum. Cases have been observed in which several tubercular intestinal strictures were present at the same time, while there was no evidence of tuberculosis in other parts of the body. Next to tubercular ulcer, the most frequent cause of cicatricial stenosis is dysenteric ulcer. Strictures of this class are located in the large intestine. Syphilis leads principally to ulcerative processes in the rectum, and in this way a syphilitic rectal stenosis, which has already been discussed, is produced. Syphilitic ulcerations with cicatricial stenosis may also occur in the upper portions of the large intestine, and, exceptionally perhaps, also in the small intestine. Typhoid ulcers only very rarely lead to cicatricial stenosis. Somewhat more frequent, it is said, are decubital ulcers that arise in the rectum from impacted feces, followed by cicatrix formation. Lastly, in isolated cases, injuries of the abdominal wall have been held responsible for causing a necrosis of a circumscribed portion of the intestine with subsequent ulcerative and cicatricial stenosis. Stenosis of the duodenum after the healing of a duodenal ulcer (*vide supra*) resembles, in its clinical symptoms, stenosis of the pylorus, and not stenosis of the intestines.

3. *Intestinal Obstruction.*—The most frequent form of intestinal obstruction comes from the impaction of feces. From the different conditions which cause enfeeblement of the peristaltic movements, an accumulation of feces (*coprostasis*) may arise, especially in the colon. This accumulation gradually but constantly increases until it leads to well-marked symptoms of intestinal obstruction, and in cases of obstruction from other anatomical causes, fecal impaction not infrequently aggravates the condition.

We see obstruction of the intestines from other causes much less frequently than from impaction of feces. In some cases impacted gallstones have been

found, especially in the lower part of the ileum and in the neighborhood of the ileocæcal valve, almost completely stopping up the lumen of the intestine. Genuine intestinal calculi (composed of phosphates) may exceptionally lead to obstruction. Likewise, occasionally conglomerations have been found, composed of indigestible portions of food, vegetable fibers, the seeds of fruit, potato skins, grape skins, and also clumps of roundworms. We must also mention here the very rare cases in which a large foreign body has been swallowed and wedged itself into some part of the intestine. Such a thing has been seen, especially in children and among the insane.

4. *Intestinal Constriction.*—Although the mechanism of intestinal constriction in external herniæ lies in the domain of surgery, we must mention here the chief causes of the so-called internal intestinal constriction (internal incarceration or strangulation). In the abdominal cavity pouches and diverticula of the peritoneum are found, as either normal or abnormal formations, in which single loops of intestine may be caught and constricted. The duodeno-jejunal hernia—the so-called Treitz's retroperitoneal hernia—is worthy of special mention, and comes from the entrance of a loop of intestine into the duodeno-jejunal fossa. This hernia may become very large. It is sometimes found by accident in the cadaver, not having caused any symptoms during life, but in rare cases it may be the cause of acute internal constriction. We must also mention the hernia of the omental bursa—where a loop of intestine passes through the foramen of Winslow—the intersigmoid hernia, the subcæcal hernia, etc. Diaphragmatic hernia is of greater practical significance because it is somewhat commoner. By this name we designate both genuine protrusions into the diaphragm and also the passage of abdominal viscera through congenital or acquired (traumatic) defects in the diaphragm. These herniæ may exist without symptoms, or at least without causing any signs of severe disease, but in some cases they cause obstruction by constricting or twisting a dislocated loop of intestine.

Those cases in which abnormal slits and holes in the omentum or mesentery give rise to internal constriction are to be added to the list of the internal herniæ. Finally, abnormal cords, membranes, and false ligaments in the abdominal cavity are a comparatively frequent cause of internal constriction. Such cords and bands are sometimes left as the results of a former peritonitis, and may cause constriction or kinks of single loops of intestine. Small diverticula of the colon sometimes form adhesions due to localized peritonitis, as we have ourselves seen, and thus occasion dangerous intestinal stenosis. One such false ligament, which must be specially mentioned, is found as a prolongation of Meckel's diverticulum. By this we mean that diverticulum which must be regarded as the remains of the omphalo-mesenteric duct, still persisting, which has its seat, corresponding to the duct, from half a meter to a meter above the ileocæcal valve. A firm cord sometimes arises from the free end of this diverticulum, the obliterated omphalo-mesenteric vein, which adheres to some part of the internal abdominal wall and may cause constriction of the intestine. Adhesion of the free end of the vermiform appendix has been the cause of internal constriction in some cases.

5. *Twists* (*Volvulus*) *and Knots of the Intestine.*—Twists about the mesenteric axis and complete constriction of a portion of intestine from this cause are seen most frequently in the sigmoid flexure, especially if the mesen-

tery of the flexure is unusually narrow congenitally. The spontaneous correction of this abnormal condition is hindered by the weight of the loops of intestine filled with gas and masses of feces, and by other portions of intestine lying on the place of twisting. Sometimes other portions of intestine wind themselves several times about the pedicle of the twisted loop so as to form a regular knot. Such twistings have been seen especially between the sigmoid flexure and a portion of the ileum. External injury sometimes gives rise to the formation of a knot. In some cases abnormally great peristalsis, severe diarrhea, precedes the appearance of obstruction. Volvulus of the small intestine is very rare, but we ourselves have observed a fatal case of volvulus in the highest part of the small intestine, consequent upon violent vomiting, excited by a remedy administered by a quack for tapeworm.

6. *Invagination of the Intestine* (*Intussusception*).—If a portion of intestine is pushed into the lumen of the portion that lies next below, we term the process invagination. The cause of this is usually assumed to be a diminution or a complete absence of peristalsis in a circumscribed portion of intestine. If now there are energetic movements in the portion immediately above, they push this into the paralyzed portion. According to Nothnagel, the beginning of the invagination is a spastic contraction of a limited portion of the intestine, in the shape of a ring. This furnishes a fixed point, and the contractions of the longitudinal muscles of the intestine below the constricted area are said to draw the lower portion of the intestine upward over this ring.

In the post-mortem examinations of children we frequently find intussusception of the ileum which has occurred shortly before death, and so is of no clinical importance. Sometimes intussusception has occasioned the most severe symptoms of intestinal obstruction. It occurs particularly in children up to the tenth year, and it may be of sudden onset, without any apparent cause. Intussusception of this sort, which often involves a considerable extent of the intestine, may have its seat at almost any part of the bowels. The intussusception most frequently involves the cæcum and the lowest part of the ileum, pushing thence into the colon (*invaginatio ileocæcalis*); intussusception of this sort may be so extensive in children that the invaginated ileum reaches into the rectum, or even projects from the anus. There are cases in which the ileum alone is involved, and less frequently the colon alone. The invaginated portion of intestine usually suffers compression of its blood vessels, particularly the veins. This is followed by inflammation of the constricted portion, and the inflammation often extends to the peritoneum. Not infrequently there is gangrene of the inner portion of the intestine, as a result of strangulation of the afferent vessels. The necrotic portions may slough off and be discharged at stool. Such an occurrence has, in a few recorded cases, led to spontaneous recovery from the intussusception and from the obstruction which it had caused.

We must mention intestinal polypi as a special cause of intussusception, as they gradually pull that portion of the intestine in which they are situated into the neighboring portion next below by their weight. This has been repeatedly observed.

7. *Compression of the Intestine from Without.*—Compression of the intestine, by tumors of the uterus, ovarian cysts, pelvic abscesses, omental

tumors, etc., has been met with in rare cases as a cause of intestinal stenosis. The symptoms of stricture in such cases develop either very gradually or sometimes rather suddenly.

Some general pathological and pathologico-anatomical conditions, which must be considered in judging all the different varieties of intestinal obstruction, have yet to be discussed.

We desire to emphasize, in the first place, that not every interference with the propulsion of the intestinal contents is necessarily due to some mechanical obstruction. Paralysis of the intestinal musculature must also produce the same result, and thus we distinguish between "a mechanical ileus"—i. e., intestinal obstruction due to an organic intestinal constriction and a "paralytic or dynamic ileus." Paralytic ileus is observed most frequently in peritonitis (*q. v.*). By an extension of the inflammatory processes from the serosa to the muscularis, or perhaps also through the toxic action of the chemical products of inflammation on the muscle fibers, a paralytic condition of the intestinal musculature, with all its sequelæ, is not infrequently produced. But conditions of this kind, which cannot be readily explained, also arise from severe injuries of the abdominal wall, and occasionally after laparotomies and herniotomies, as well as after severe gallstone and renal colics. The natural thought is of a reflex inhibition of the intestinal peristalsis. The rarer cases of paralytic ileus observed after embolism of the mesenteric artery are more easily understood. In addition to the paralytic ileus, we also speak of a spastic ileus (in hysteria, lead poisoning); but one must be very careful in the interpretation of such conditions.

True mechanical ileus is also divided into two important groups—the simple obturation, or occlusion ileus, and the strangulation ileus. The importance of this distinction depends on the fact that the condition of the intestinal wall is entirely different in these two forms of intestinal obstruction. In occlusion of the intestine (from coprostasis, gallstones, etc.) the nutrition of the intestinal wall is not primarily affected, whereas in strangulation, by compression of the afferent and efferent blood vessels, an arterial anæmia, or venous hyperæmia, is produced in the strangulated (or invaginated) piece of gut. In this way the nutrition of the intestine is disturbed. The intestine becomes permeable to bacteria, it is distended with gas, and may finally become necrotic and gangrenous (*vide infra*).

The condition of the intestine above the constricted portion deserves particular attention. The coils of intestine above this part are generally distended with gas and accumulation of feces. But it should be noted that the abundant fluid found above the narrow part of the intestine is by no means due exclusively to the ingesta, but is largely the result of transudation and the secretion of intestinal juices. The intestinal contents are very apt to undergo putrid decomposition. This gives rise to a large amount of gas which may not be reabsorbed, so that above the obstruction there is apt to be a very marked tympanites.

In simple obturation stenosis the meteorism extends almost over the entire portion of intestine situated above the constricted part (meteorism due to back pressure).

In stenosis due to strangulation (*vide supra*), however, the meteorism is

confined at first to the constricted loop of intestine with impaired circulation (local meteorism). The local meteorism is followed only later by a general passive tympanitic distention of the intestines. All intestinal coils which are tympanitically distended soon suffer marked lesions of their walls. In passive meteorism the distention of the abdominal wall and the associated disturbance in circulation must be considered, but in addition, also, the irritation of the decomposing intestinal contents, as well as the purely mechanical pressure of the stagnating fecal masses. With local meteorism, the circulation of the constricted loop of intestine is interfered with from the start. We can therefore readily understand that inflammatory changes, which may progress to diphtheria and circumscribed necrosis, set in above the stenosed portion, and also, with great rapidity, in the constricted loop of intestine. Diseased intestinal walls of this kind are abnormally permeable to bacteria and toxins, even if no actual tear or perforation has occurred. Pyogenic bacteria or putrified intestinal contents thus pass readily into the abdominal cavity, and the onset of a severe purulent or gangrenous peritonitis is unavoidable. This is why acute peritonitis is so frequent a lesion in persons who die of intestinal obstruction.

If the intestinal stenosis has lasted a long time, we usually find in the upper portion of the intestine, besides the signs of inflammation, a manifest hypertrophy of the muscular coat, the result of the abnormally active peristalsis by which the muscle has tried to overcome the obstacle. The intestine below the constriction, in contrast to the part just described, appears narrow, contracted, and empty.

Clinical History.—We must distinguish the cases with a rapid, complete obstruction of the intestine from those in which the condition develops gradually, and where there is, therefore, at least for a time, merely a constriction of the intestine.

1. *Intestinal Constriction.*—The first symptom of intestinal constrictions that occur, particularly in cancer of the intestines and cicatricial strictures, and more rarely in constrictions from chronic peritoneal adhesions and bands, or from compression of the gut from without, etc., are disturbances of defecation. The bowels are costive, they move only at long intervals, and their motion is often associated with pain and tenesmus. In the description of cancer of the intestines we have already mentioned that the feces passed sometimes have a peculiar, flat, compressed, or scybalous form.

But since stools of this nature also occur under other conditions (obstinate chronic constipation, insufficient nourishment), and may occasionally be absent in cases of existing stenosis, their diagnostic significance should not be overestimated.

Blood and mucus are often mixed with the dejections and are due to the character of the primary disease. In some cases there is no constipation, and there may be even constant diarrhea, due to a catarrh of the intestinal mucous membrane above the stenosis (*vide supra*). We can easily understand from the physiological conditions that in stenosis of the small intestines, whose contents have an approximately fluid consistence, disturbances of defecation are less apt to take place than in stenosis of the large intestine, where the fecal masses have already assumed a more firm consistence.

Physical examination of the abdomen often gives important and valuable

information. The abdomen is usually swollen by meteorism (*vide supra*). The intensity of the meteorism varies very much in different cases and at different times in the same patient. Meteorism is sometimes absent, especially in stenosis at the beginning of the intestine. There may then be gastrectasis. The marked peristaltic movements, plainly visible through the abdominal walls, are very characteristic of most intestinal contractions. The contour of single loops of intestine is often marked, at times quite sharply, and then we can sometimes feel the thickened intestinal walls through the lax abdominal wall (the so-called "intestinal erection" of Nothnagel). We may often decide upon the seat of the stenosis from the location and course of the visible peristaltic movements. In general, it is true that peristalsis is much more noticeable when the stenosis is in the small intestine than when it is in the colon.

In most cases of intestinal stenosis severe colic attacks frequently accompany the visible intestinal contractions. The point of origin of the pains, at least in many cases, corresponds approximately to the site of the stenosis.

If we put our ear to the anterior abdominal wall we can often hear many gurgling and splashing noises, which sometimes have a distinct metallic quality. Eructations are frequent, and in some cases there is occasional vomiting. We must finally state that we have been repeatedly struck by the great extent and strength with which we could feel the pulsation of the aorta through the swollen loops of intestine.

The duration of all these symptoms varies with the nature of the primary lesion. Of course the greatest importance attaches to the condition of the muscular layer of the intestine above the constricted spot. For a time the muscle may overcome the obstruction because of its gradual hypertrophy, but at last the muscular coat becomes insufficient, and consequently the symptoms become more severe. This explains why the symptoms of intestinal stenosis change gradually or suddenly into those of complete intestinal obstruction. With this change we have grave symptoms. Sometimes, in cases of chronic stenosis of the intestine, some temporary causes may occasion repeated attacks of intestinal obstruction, which, however, are relieved and give place again to the milder symptoms of mere stenosis.

2. *Intestinal Obstruction.*—The symptoms of intestinal obstruction (*ileus miserere*) either develop gradually from the preceding symptoms of intestinal constriction, or occur at the outset in all their menacing severity. But here, also, there are many variations, according to the nature of the morbid process which is the cause of the intestinal obstruction. For the present we will disregard ileus following a preceding peritonitis (paralytic ileus, *vide supra*); we can then divide the remaining varieties, as previously stated, into two main groups. In the one group (obturation or occlusion ileus) we have to deal at first more with a mechanical obstruction of the lumen of the intestines and its consequences, while at first the intestinal wall itself is not damaged (at least not in its entirety). To this category belong intestinal occlusions from coprostasis, from gallstones, and as a sequel to preceding stenosis caused by tumors and cicatrized ulcers, from compression from without, etc. The second group, on the other hand, is designated as strangulation ileus; here the loop of intestine, which is either squeezed, twisted about its pedicle, or incarcerated, suffers a grave disturbance in its circulation from the very start, and, in consequence,

soon becomes inflamed and permeable to bacteria and their toxins. These are the cases in which the purely mechanical symptoms of intestinal obstruction are soon followed by diffuse septic peritonitis.

The fully developed clinical picture of ileus generally permits us to recognize the gravity of the condition at a glance. The face of the patient shows a well-marked "facies abdominalis," the eyes and cheeks are sunken, the nose is pointed, and the entire face, as well as the extremities, cool and cyanosed. The voice is feeble and weak, but the intellect is perfectly clear. The tongue is dry, respiration is difficult, as the diaphragm is displaced upward the pulse becomes rapid and small, and, finally, scarcely perceptible. The temperature is usually subnormal, but elevations of temperature may occur if peritonitis has set in. Movements of the bowel and expulsion of gas cease entirely, often in spite of a desire to defecate. On the other hand, eructations and vomiting soon set in. The latter may, at first, be a simple reflex vomiting, such as is observed particularly in acute strangulation. If, however, the intestinal occlusion lasts for some time, the vomiting soon assumes a fecal character ("stercoraceous vomiting"), which is quite characteristic of ileus. The odor of the vomitus becomes more and more foul and distinctly fecal, and finally completely resembles diarrheal movements. As has been previously mentioned, we are dealing here with a putrefaction of stagnating intestinal contents above the site of obstruction. It is possible that these masses are conveyed upward into the stomach by antiperistalsis, but this statement is disputed by many observers. In the main, it appears to be simply a damming back and regurgitation, practically an "overflow," of the filled intestinal loops, into the stomach, since the pylorus gradually gives way to the increasing distention of the small intestines.

The examination of the abdomen is of the greatest importance. There is generally tympanitic distention of the abdomen, but the meteorism varies somewhat in the different cases. If the stenosis is situated low down in the large intestine, the colon becomes distended first. Corresponding to its anatomical position, it becomes more or less clearly defined as a thick, long, rounded prominence. In obstruction of the small intestine, on the other hand, the meteorism is more pronounced in the middle part of the abdomen. The distinction above drawn between general meteorism from stagnation and localized meteorism is of especial importance. By exact comparative palpation during the initial stage in acute strangulation we can occasionally locate the circumscribed distention of the strangulated intestinal loop. This inflated and fixed strangulated coil shows no peristalsis, and remains entirely immobile. Occasionally, not the strangulated loop, but the one situated above the site of strangulation, becomes prominent through a damming back of the tympanitic distention. Moderate peristaltic motions and "erections" can be produced in it by slight mechanical stimuli. The distention becomes more general and more pronounced with the increase in the symptoms, until finally, with the onset of peritonitis, general paralysis of the intestines sets in. The percutory conditions depend on the degree of gaseous distention of the intestines and the amount of exudation that, under certain conditions, forms in the abdominal cavity. An initial severe abdominal pain occurs especially in acute strangulation of the intestines. Later on there is a more continuous pain or else acute exacerbations, which can rarely be sharply localized. In fatal cases the pains frequently cease entirely with the onset of general intestinal paralysis. Pres-

sure on the abdomen in intestinal obstruction is, as a rule, not painful; it may even slightly diminish the pain. The abdomen becomes very tender only after peritonitis has set in.

The excretion of urine, especially in cases of obstruction in the small intestine, is generally diminished, on account of the decreased absorption and the general desiccation of the body, through the exudation into the intestine or the peritoneum. Not infrequently the scanty urine contains albumen, casts, and blood corpuscles. The increase of indican [1] in the urine is also important. In the stagnating intestinal contents above the stenosis large quantities of indol, phenol, etc., are produced by putrefaction of the proteid materials. Indol is excreted as indican. Marked indicanuria is found principally in stenosis of the small intestine, because proteid bodies, capable of decomposition, generally occur only in small amounts in the large intestine. The so-called Rosenbach's reaction (a deep-red coloration with a violet foam, on adding nitric acid drop by drop to the boiling urine) is also occasionally found in severe cases of intestinal strangulation.

The general course and duration of intestinal obstruction naturally varies greatly with the nature of the underlying morbid process. We have already emphasized the practically important distinction between intestinal occlusion, which generally takes a slower course, and only later on gives rise to the grave clinical picture of ileus with general collapse and peritoneal symptoms, and the clinical picture of intestinal strangulation, which much more rapidly reaches an alarming degree of severity. The gravest cases may end fatally in two to three days. The duration of the disease is generally about a week. In cases of simple obstruction, or those which develop only gradually from a stenosis, the duration of the illness may be very much more prolonged. The danger consists, above all, in a poisoning of the body through absorption of the products of putrefaction and a consequent vasomotor and cardiac paralysis, and, furthermore, in the onset of secondary peritonitis. The majority of the serious cases end fatally.

Aside from operative interference, recovery may ensue even after the gravest symptoms, but it is rare. Cases of intestinal obstruction by obturation are the most likely to recover. Incarcerated gallstones, impacted feces, etc., may be discharged with a subsidence of the grave symptoms. The possibility of recovery by sloughing off of the gangrenous internal portion of intestine in cases of invagination has already been alluded to above. But it is undoubtedly possible that cases of internal strangulation also may be spontaneously relieved so long as no serious nutritive disturbances have occurred in the strangulated loop.

As soon as general peritonitis has set in recovery is impossible. However, on account of the difficulty of an accurate diagnosis, the prognosis of the individual case is often uncertain.

Diagnosis. Varieties of Intestinal Obstruction.—To go into details as to the clinical symptoms of all the separate forms of intestinal constriction and obstruction would lead merely to repetitions. Our judgment of each case is

[1] The indican test is performed in the following way: We mix equal volumes of urine and officinal hydrochloric acid (P. G.), and then add, drop by drop, a concentrated solution of chlorid of lime. If, now, chloroform is added, upon shaking carefully the chloroform will take on a very distinct blue color if the urine contains any considerable amount of indican.

based, in the first place, on a very careful history, which often furnishes material diagnostic aid (gallstones and other previous diseases, rapid or slow onset, etc.). In the objective examination that then follows, all external hernial canals are examined first (inguinal and femoral canals, umbilicus). Thereupon there should be a careful inspection and palpation of the abdomen, in which particular attention must be paid to a circumscribed distention of individual loops of intestine, visible peristalsis, localized points of tenderness, etc. Finally, rectal and vaginal examination should never be omitted.

Schematically arranged, the distinction between obstruction and strangulation is based principally on the following points:

OBSTRUCTION	STRANGULATION
No symptoms of collapse at first. These develop only later on, and gradually. Pulse remains strong for a long time. Marked pain is rare at the onset.	At the onset grave symptoms of collapse and facies abdominalis. Small rapid pulse. Frequently violent initial pain, which continues.
Vomiting occurs late (overflow vomiting).	Initial (reflex) vomiting. Later on, also overflow vomiting.
Meteorism becomes rapidly diffuse.	Localized meteorism at first in the strangulated loop of intestine. Later on, also meteorism due to back pressure.
Exudation into the abdominal cavity is generally absent.	Slight exudation into the abdominal cavity frequently occurs from the strangulated piece of intestine.

With regard to distinguishing between stenosis of the colon and of the small intestine, stenosis of the small intestine is indicated by a special prominence of the central portion of the abdomen, by the visible and active peristaltic motion of many coils of intestine, by the occurrence of fecal vomiting, and by the presence of a strong reaction of indican in the urine; while stenosis of the large intestine is characterized by a distention which corresponds more with the course of the colon, without much visible peristalsis, and a slow development of severe constitutional symptoms. In a few cases, inflation of the large intestine with air is said to have cleared up the diagnosis where the stenosis was situated low down. Important data are occasionally obtained by X-ray examination. Certain deductions may occasionally be made from the amount of fluid which can be introduced into the rectum. We shall recur later to the differential diagnosis between intestinal obstruction and diffuse peritonitis, when considering the latter.

Of the special varieties of intestinal obstruction, we will call attention to three as of practical importance—viz., intussusception in children, volvulus of the sigmoid flexure, and fecal impaction.

Intussusception appears, as we have indicated, mainly in children under ten years. Its symptoms usually begin rather suddenly with violent colicky, abdominal pain. There soon appear, also, liquid dejections of bloody mucus

ACUTE INTESTINAL OBSTRUCTION

CHIEF COMMON SYMPTOMS.—Sudden *pain*, intermittent or constant, with exacerbations; tends to become constant with time. *Vomiting*, early, severe, becoming feculent. *Constipation*, more or less absolute. Abdominal *distention*. *Shock*.

	STRANGULATION BY BANDS OR THROUGH APERTURES (25 *per cent of all cases of acute obstruction*).	VOLVULUS OF COLON.	ACUTE INTUSSUSCEPTION.
AGE AND SEX...	Young adults; rare after 40.	Males at 4 : 1 : 40 to 60.	More than 50 per cent under 10 years.
HISTORY.....	Previous peritonitis in 68 per cent; previous attacks of obstruction in 12 per cent.	Previous constipation.	Usually negative.
ONSET.....	Sudden in 70 per cent.	Sudden.	Sudden in 75 per cent.
PAIN.....	Early, severe, continuous, with exacerbations.....	Early, less severe, intermittent at first, becoming constant with exacerbations.	Early and severe; increasing and later subsiding; at first paroxysmal.
LOCAL TENDERNESS.....	Absent at first, appears later.	Early over distended coil, and constant.	Common about a tumor.
VOMITING.....	Early, marked; in 60 per cent becomes feculent; affords no relief.	Less early, severe and constant; often affords relief.	Still less early and severe; in 25 per cent becomes feculent.
CONSTIPATION..	Continuous and absolute; no blood.	Early and absolute; no blood.	Seldom absolute; diarrhea not uncommon; blood in 80 per cent.
PROSTRATION...	Marked.	Rather less marked; may be dyspnœa.	Marked.
TENESMUS.....	Absent.	In 15 per cent.	In 55 per cent and often early.
ABDOMINAL WALL.....	Flaccid unless peritonitis.	Rigid from early peritonitis.	Flaccid unless peritonitis.
TUMOR.....	Very rare.	Absent.	In 50 per cent; invagination sometimes felt in rectum.
METEORISM....	Slight, appears about third day.	Early, rapid, increases, and is extreme.	Rare, unless marked constipation.

N. B.—No trustworthy conclusions can be drawn from the seat of the pain as to the seat of the obstruction unless local peritonitis comes on. The pain is usually referred in all forms to the region of the navel. In complete obstruction the pain is constant though with exacerbations; intermittent pain shows that the obstruction is partial. Coils of intestine are not visible through the abdominal wall in acute cases.

or almost clear blood, originating from the constricted portion of the intestine. Often there is great tenesmus and protrusion of the anus. We have already mentioned that sometimes the invaginated portion of intestine may be felt in the rectum. The sausage-like tumor of the invaginated portion of intestine can occasionally be felt on external palpation along the course of the colon.

The further course corresponds with that of intestinal obstruction, except that it should be said that fecal vomiting generally occurs in the most frequently observed ileocæcal intussusception only comparatively late in the attack.

Volvulus of the sigmoid flexure generally occurs in older people who have previously suffered from chronic constipation. The onset of these grave symptoms is very sudden. A severe pain is felt in the left iliac fossa. The visible and palpable distended intestinal loop extends from this region upward toward the diaphragm. General meteorism soon supervenes. Small quantities of blood are occasionally passed by rectum. Stercoraceous vomiting occurs only late, if at all. The amount of fluid which can be introduced into the rectum is small. The remaining symptoms correspond to the general picture of an internal strangulated hernia.

CHRONIC INTESTINAL OBSTRUCTION

	Stricture of the Small Gut.	Stricture of the Large Gut.	Fecal Accumulation.
Age and Sex...	Adults.	Adults.	Adults; more common in females, the hysterical, the insane, hypochondriacs.
History.....	Cancer, trauma, tuberculosis; disordered, imperfect, irregular action of bowels from time to time, with intervals of comparative ease.	Cancer, trauma, tuberculosis, dysentery; disordered, imperfect, irregular action of bowels from time to time, with intervals of comparative ease.	Previous constipation.
Onset.....	Gradual.	Gradual.	Gradual.
Pain.....	Intermittent.	Intermittent.	Less prominent.
Vomiting.....	Late, scanty, feculent only toward end of acute attack; may be provoked by food.	Less prominent, rarely feculent or provoked by food.	Late, scanty, rarely feculent, often absent.
Constipation..	May alternate with diarrhea; blood points to cancer.	Form of feces may be altered; blood points to cancer.	Gradually increasing; may be spurious diarrhea; no blood.
Tenesmus.....	Absent.	Often present.	Absent.
Meteorism....	Not marked, unless acute attack.	Often marked.	Late; generally increases with obstruction.
Tumor.....	Only in cancer, and then in 30 per cent.	Only in cancer, and then in 40 per cent; may be felt in rectum.	Common and distinctive; most easily felt in cæcum; little or no tenderness; sometimes movable, and can be changed in shape.
Coils of Intestine.....	Marked in proportion to emaciation.	Marked in proportion to emaciation.	Rarely seen.

N. B.—In any form of chronic obstruction, the symptoms of acute occlusion may suddenly supervene.

Lastly, we desire to mention, as of practical importance, that form of intestinal obstruction which is produced by the accumulation of large quantities of old fecal masses in the rectum. We sometimes find monstrous accumulations of feces in the rectum, especially in old women who have previously suffered from habitual constipation, or in whom constipation is due to some other affection. Severe symptoms usually come on quite suddenly, after long-continued mild prodromal symptoms, and these severe symptoms are much like the picture of internal strangulation—severe, sometimes colicky, abdominal pain, great tenderness of the abdomen, which is usually swollen, marked general collapse, loss of strength, a small pulse, an outbreak of cold sweat, vomiting, etc. If we try to give an enema in such cases, very little fluid runs into the rectum. On introducing the finger, it usually strikes solidly on old, hard, fecal masses above the sphincter, and there is often nothing left but to undertake the dirty task of removing at least a part of the scybala with our own hands. We may then succeed, by repeated enemata and by giving cathartics internally, in removing sometimes quite an incredible amount of accumulated feces, and in obtaining thus a rapid recovery from the condition.

The diagnosis of the nature of an intestinal obstruction is so difficult in many cases, and yet so important with reference to treatment, that the editor ventures to introduce tables of differential diagnosis of the more common forms of the condition. These tables are based upon the masterly prize essay of Treves of London.

Treatment.—As soon as the dangerous signs of intestinal obstruction are recognized by the physician, his first duty is to make as careful an examination (*vide supra*) as possible, so as to determine whether the obstruction may not be within the reach of direct treatment. If there is an incarcerated external hernia, it demands special surgical treatment. If there is an impaction of gallstones, or intestinal calculi, or foreign bodies, we may in some cases furnish appropriate assistance by the cautious use of laxatives. The treatment of fecal impaction is of special importance. We have described the most frequent form of this in detail above. As has already been said, it is usually necessary to remove at least a part of the feces with the fingers, or some instrument like a dressing forceps or a spoon. In the second place, we may use large enemata of pure water, or soapsuds, or oil, which must often be repeated four or five times a day, until they have a satisfactory result. These are best given by a funnel and an œsophageal tube ("intestinal tube") introduced as high as possible into the intestine. Cathartics administered internally serve as aids, especially castor oil, senna, saline cathartics, etc.

In stenosis of the rectum from cicatrices and new growths we can also sometimes employ local surgical treatment. The treatment of fecal accumulations usually plays an important part here. Finally, the cases of ileocæcal invagination, in which the lower end of the invaginated ileum reaches the rectum, may receive local treatment. We may try a partial replacement by a "sponge sound" (an elastic œsophageal tube to the end of which a sponge is fastened). Blowing in air by the bellows was also recommended for this purpose by the old physicians. As a rule, however, we use here large enemata of warm water, which sometimes seem to exert a favorable mechanical action.

If the symptoms of a more or less chronic constriction of the intestinal lumen exist, the treatment consists, at first, only of a regulation of the diet (a fluid or soft diet, which produces only little fecal matter), mild cathartics, and the occasional administration of opium, perhaps with belladonna, where the peristalsis is increased and painful. Externally, cold packs or warm applications are generally used. Above all, it is important to decide the question whether an operative removal of the obstruction, or at least palliative surgical treatment (an artificial anus), is possible.

In acute intestinal obstruction we must also, first of all, consider whether timely surgical intervention may still bring relief. The more the symptoms, grave from the onset, point to an acute intestinal obstruction, the more urgently should early laparotomy be advocated. If the symptoms are less fulminant, and the general condition good, expectant treatment may be pursued at first. But here, also, we must caution against delaying too long.

If we cannot decide on an operation, or if the latter is impossible, internal symptomatic treatment of the ileus is then in order. It goes without saying that the patient should have complete physical rest. All food should, if possible, be entirely forbidden. The torturing thirst may be relieved by bits of ice or sips of cold bitter tea. Formerly when there was obstruction of the bowels, it was the custom to make trial of laxatives, first employing milder remedies, then more vigorous drugs, and finally, as a "last resort," metallic mercury in single doses of from 5 to 10 ounces (gm. 150 to 300), which is sometimes claimed to act mechanically in "desperate cases" by its weight. Except among some champions of mercury, the present opinion among physicians

tends far more to the belief that cathartics are usually of no service, but are often directly injurious by increasing the resistance. Hence we have at present gone over to the treatment of severe internal incarcerations with large doses of opium (20 drops of laudanum or 2 to 3 gr. [gm. 0.1 to 0.2] of opium, several times a day). It is often very advantageous to administer opium in suppositories (ext. opii, gr. j to ij = gm. 0.05 to 0.1). Opium acts favorably on the patient's pain, the vomiting is diminished, and, by quieting the peristalsis, the danger of increasing the stenosis and tearing the intestine is also lessened. Sometimes the first dejection appears during the administration of opium. Morphin subcutaneously injected has much less effect upon the intestine than opium; its employment is therefore limited to cases in which pain is so violent as to demand relief. Objection is made to the opium treatment, not without some justice, that by lessening the pains it obscures the gravity of the disease. This objection is, however, justified only if opium is used without strict indications and haphazard. Besides opium, atropin has recently been repeatedly strongly recommended. It seems doubtful if it is efficacious in true strangulative ileus. In intestinal obstruction, and perhaps also in the paralytic form of ileus, its usefulness appears to be more probable, since in small doses (gr. $\frac{1}{120}$ to $\frac{1}{60}$ = gm. 0.0005 to 0.001) it stimulates intestinal peristalsis. In some cases, good results are said to have been obtained only after larger doses (gr. $\frac{1}{60}$ to $\frac{1}{30}$ = gm. 0.001 to 0.002).

Although, as already mentioned, there are a number of objections to the internal use of cathartics, we may try large enemata in many cases. The injections must be given cautiously but persistently, and they must be often repeated. They sometimes give good results even in severe cases.

Instead of plain water, we generally use soapsuds, and occasionally, also, oil injections. Unpleasant effects have been reported by other observers, but we ourselves have never seen any. The introduction of air into the rectum, instead of water, has been recommended and has been useful, particularly when the obstruction was situated low down and was the result of a kink or some similar trouble. The employment of regular gastric lavage (Kussmaul, Cahn, and others) is very beneficial in many cases of fecal vomiting; if this is a prominent symptom, the stomach should be washed out in every case. Large amounts of feculent liquid are frequently removed through the stomach tube, and it is easy to see that freeing the stomach of its accumulations may favor a more vigorous peristalsis. Even when the nature of the intestinal obstruction precludes definitive recovery, lavage usually gives no inconsiderable relief.

We need not enter into details as to the general treatment. It goes without saying that the patient's strength must be kept up as much as possible, and that in severe stages of collapse all possible stimulants must be used, such as strophanthus, camphor, and ether. Local applications to the abdomen are usually ill borne on account of the tenderness. Wet compresses may be tried, or, what is still better, hot moist packs applied to the entire abdomen. Opium is the best remedy for pain and vomiting, as already stated, but it must often be replaced by subcutaneous injections of morphin. In cases of extreme gaseous distention, the attempt has occasionally been made to puncture the distended intestinal coils with a Pravaz syringe, in order to partially evacuate the gas. This method appears, however, to be not entirely devoid of danger.

[The safety with which laparotomy is now performed has stimulated the study of all affections on which the operation has any bearing. Internal strangulations and invaginations may be relieved, and the portion of intestine containing a noncancerous stricture can be excised. An early operation offers much better chances, of course. In these days persons should not be allowed to die directly from intestinal occlusion without an attempt being made to restore the permeability of the canal by surgical means.]

CHAPTER XII

INTESTINAL PARASITES

(Helminthiasis)

1. TAPEWORMS

Natural History of the Tapeworm.—Three of the tapeworms (*cestodes*) which are found in the intestines have a clinical significance: the *tænia solium*, the *tænia mediocanellata*, and the *bothriocephalus latus*.

1. The *tænia solium* is, when fully developed, 2 or 3 meters long. Its head (Figs. 74 and 75) is about the size of that of a pin, and has four projecting cup-like suckers, and in front a beak with about twenty-six hooks. The top of the head is, as a rule, plainly pigmented. A small neck, about an inch (2 to 3 cm.) long, is attached to the head, and then follow the single

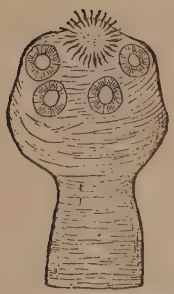


FIG. 74.—(From HELLER.) Head of tænia solium.

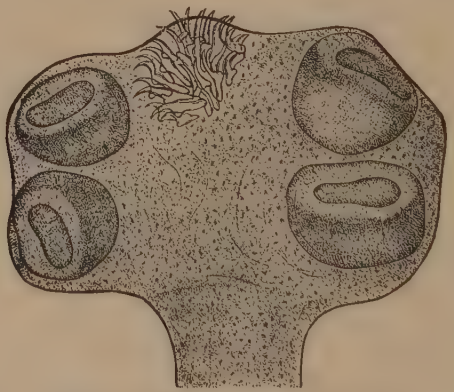


FIG. 75.—(From HELLER.) Head of cysticercus of the brain.

FIG. 76.—(From HELLER.) Tænia solium. Mature segment.

"segments" (proglottides) of the tapeworm, of which the youngest, lying near the head, are still very small and short. They gradually increase in length and breadth, and at about a meter from the head they have an approximately quadrilateral shape. The segments, which lie farther down, and which have already reached puberty, have the form of pumpkin seeds, and are $\frac{1}{3}$ to $\frac{2}{5}$ of an inch [9 or 10 mm.] long and 6 or 7 wide. The matrix or uterus runs through the middle of each mature segment (see Fig. 76), and from it, on each side, go seven or eight side branches, which ramify like a tree. On one side, a

little below the middle, lies the sexual orifice (Fig. 76, *a*). The male sexual organs consist of a number of little clear vesicles in the anterior portion of the segments. The thick-shelled eggs (Fig. 77, 3) develop in the uterus, and contain an embryo with six hooklets.

The tænia solium inhabits the small intestines of man. Its head clings to the mucous membrane so tightly, usually at some point in the upper third

FIG. 77.—Comparative view of the eggs of some of the commoner intestinal parasites. 1. Egg of distoma hepaticum. 2. Distoma lanceolatum. 3. Tænia solium. 4. Tænia mediocanellata. 5. Bothriocephalus latus. 6. Oxyuris vermicularis. 7. Trichocephalus dispar. 8. Ascaris lumbricoides.

of the small intestine, that the neck is often torn off in trying to loosen the worm from the intestinal wall. The rest of the worm, which is in part in many coils, extends to the lower part of the ileum, but only exceptionally into the cæcum. From the lower end long chains, only rarely single mature segments, are detached, mix with the contents of the intestine, and are passed with the feces, together with some of the eggs from the uterus.

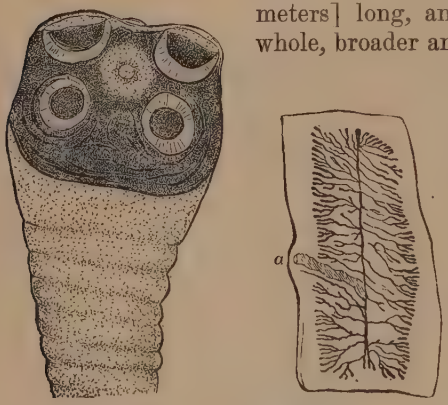
The further development of the eggs of the tænia solium takes place in another "host," almost always in the hog. Hogs are infected by eating feces, offal, etc., containing tænia eggs. The thick shell of the eggs is dissolved in the hog's stomach, and the free embryos pierce the walls of the stomach and intestines and travel with the blood current, or through the tissues, into the different organs, especially into the muscles. Here they develop, in two or three months, into cysts something larger than a pea, from whose walls a newly developed tænia head arises, a so-called *scolex* (nurse). These cysts are termed worm cysts, measles, or *cysticerci cellulosæ*. They live from three to six years; then they die and become calcified. If a cysticercus gets into a man's stomach from his eating raw or imperfectly cooked ham or pork, a new and complete tænia sprouts from the scolex, which forms mature segments in three or four months.

We usually find only one tapeworm in a man, but several specimens have been seen (as many as seventeen) at the same time in the same intes-

tine. The length of a tapeworm's life is not certainly known, but it has happened that some persons have lodged the same tapeworm for ten or fifteen years.

Although the fully developed tænia solium is seen only in man, as we have said, the cysticercus cellulosæ has been found, in rare cases, in dogs, rats, and monkeys, etc., as well as in hogs. It is a particularly important fact that the cysticercus cellulosæ itself may also occur, as such, in man. If tapeworms or mature segments get into a man's stomach in any way, probably by autoinfection by the finger, etc., the embryos travel into other organs. Cysticerci are often found in men, singly or in groups, especially in the skin, the brain, the eye, and the muscles. There is a special form of cysticercus of the brain, in which we find a whole chain of cysts, like a cluster of grapes, but sterile, the so-called cysticercus racemosus.

2. The *tænia mediocanellata*, or *tænia sagnata* (from *saginare*, to fatten), is far more common than the tænia solium in many parts of Germany. It is longer than the tænia solium, being about 10 to 13 feet [3 or 4 meters] long, and its individual segments are, on the whole, broader and thicker. The head (Fig. 78) has also four prominent cup-like suckers, but it has no crown of hooklets. The mature segments differ from the proglottides of tænia solium, in that the central uterus sends off many more (twenty to thirty) side branches, which divide dichotomously, and not like a tree. The sexual opening is also on the side (Fig. 79, *a*).

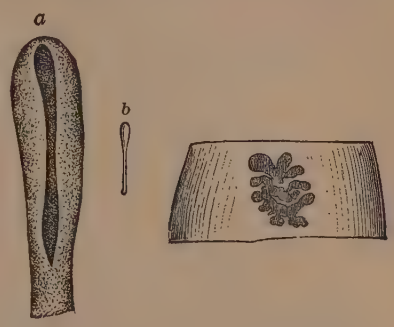


Figs. 78 and 79.—(From Heller.)
Fig. 78.—Head of tænia mediocanellata.
Fig. 79.—Tænia mediocanellata. Mature segment.

The life-history of the tænia mediocanellata is, on the whole, like that of the tænia solium. The tænia mediocanellata, however, throws off single mature segments much more frequently than the tænia solium. These segments are found in the feces, and here they often exhibit a crawling motion. A kindred fact is that it is always easy to find large numbers of tapeworm eggs in the feces of individuals who harbor the tænia mediocanellata. This is usually difficult in the case of tænia solium. The eggs of tænia mediocanellata and tænia solium are very similar (*vide* Fig. 77). They have a tough, radially striated shell, and in both varieties the six hooks of the embryo can be seen in the interior. The cysticercus of tænia mediocanellata does not inhabit pork, but beef, so that the infection of man by this tapeworm comes from eating raw beef. In man the cysticercus of tænia mediocanellata, which is somewhat smaller than the cysticercus cellulosæ, has never yet been observed.

3. The *bothriocephalus latus* occurs in Holland, Switzerland (Geneva), Pomerania, East Prussia, Hamburg, and Russia (the German Baltic provinces). It has been observed in middle Germany only in isolated cases. It is the largest tapeworm; it may be 6 or 8 meters long, and sometimes has over 4,000 joints. The head of the bothriocephalus (Fig. 80) consists of a little club-shaped

swelling, with two slit-like depressed suckers on the sides. A long, thread-like neck joins the head to the youngest segments. The full-grown segments (Fig. 81) are short, but are distinguished by their great breadth. The largest segments measure in length about $\frac{1}{8}$ to $\frac{1}{6}$ in. [3 or 4 mm.], and in breadth $\frac{2}{5}$ to $\frac{1}{2}$ in. [10 or 12 mm.], but the last joints are longer and are not so broad, so that they have an approximately quadrilateral form. The uterus consists of a very tortuous canal in the center. The sexual orifice does not lie on one side, as in the tænia, but in the middle of the abdominal surface, nearer the anterior border of the segment than the posterior. The eggs (*vide supra,* Fig. 77, 5) are of an oval form, and have a hood-shaped lid at one end. They are to be found in almost every dejection of persons affected with a bothriocephalus. Single joints of the tapeworm are not passed with the stools, but portions of the worm, several feet long, come away from time to time, especially in the spring and autumn.



FIGS. 80 and 81.—(From HELLER.)

FIG. 80.—Head of bothriocephalus latus. *a*. Lateral view, enlarged. *b*. Natural size.

FIG. 81.—Bothriocephalus latus. Mature segment.

The eggs develop only in fresh water. The embryo (Fig. 82), which is formed in them in a few months, and is provided with six hooklets and with vibrating cilia, is swallowed by fishes (chiefly pike and eel-pouts, according to Braun, salmon, according to Küchenmeister), and develops in their muscles and internal organs into cysticerci. The infection of man with bothriocephalus comes from eating such fish containing cysticerci.

FIG. 82. — Embryo of bothriocephalus latus, with its ciliated coat. (LEUCKART.)

[In the United States, the tænia solium is sometimes seen, the tænia mediocanellata is common, and the bothriocephalus is practically confined to immigrants.]

4. These three kinds of tapeworms that have been enumerated are the most important, but we will briefly mention the *tænia nana* and the *tænia cucumerina* (*tænia elliptica*). The first of these, the tænia nana [dwarf], is the smallest tapeworm, being only $\frac{2}{5}$ to $\frac{3}{5}$ in. [1 to 1.5 cm.] long. The long head is provided with four suckers and a circlet of hooks which can be extended far out. The anterior joints are very narrow and short, those farther back broadening not inconsiderably. The tænia nana is very frequent in Italy and also in Sicily. In Germany it has been seen only exceptionally. Its cysticercus is said to inhabit snails. It is seen especially in children, and in them sometimes in very great numbers. Single joints are difficult to find in the dejecta, but the discovery of its eggs with the microscope is very easy. The expulsion of the worm by means of santonin and thymol (*vide infra*) is usually a simple matter.

The tænia cucumerina is 8 to 10 in. [20 to 25 cm.] long. Its posterior joints are 6 to 8 mm. long, but have a width of only 1 mm. The head is provided with many hooklets; the beak can be extended and retracted. The tænia cucumerina occurs especially in children. Its cysticercus is found in the dog.

Symptoms and Diagnosis.—In many cases tapeworms are lodged in the intestines without causing any morbid symptoms. We can recognize their presence only by occasionally finding the segments in the dejections.

In other cases, however, tapeworms cause a list of disturbances which are often exaggerated by anxious, hypochondriacal, and nervous persons, but which ought not to be too little regarded. The symptoms are referred chiefly to the intestinal canal. Sometimes there is quite severe abdominal pain, which may assume a colicky character. The patient also frequently complains of irregularity of the bowels, and of occasional diarrhea, which alternates with constipation. Many general symptoms are also added to those mentioned—loss of appetite, or at times marked veracity, general languor, disinclination to work, mental disturbance, depression, etc. Often the general nutrition is considerably impaired.

There are also certain symptoms to be mentioned which were formerly explained as "reflex." Still, it is much more probable that poisonous matter may be produced by the tapeworms under certain circumstances, about which we do not yet have accurate knowledge, and that these poisons occasion some of the symptoms of tapeworm (*vide infra*). Among these we sometimes see marked salivation, tickling in the nose, dilatation of the pupils, palpitation, vomiting, headache (migraine), etc. In some cases even severe spasms and choreic conditions have been referred to the presence of tapeworms (particularly the tænia nana) in the intestinal canal, but it is hard to decide how far such a supposed connection can really be regarded as justified.

The peculiar fact that nearly all cases of helminthiasis lead to a marked eosinophilia (increase of the eosinophile leucocytes) of the blood, must also be referred to the production of certain substances.

Thus, in most cases tapeworms cause comparatively little disturbance, but they may occasion severe illness. It must especially be pointed out that when the bothriocephalus latus, or exceptionally a tænia, is present in the intestines there will often be an extremely severe anæmia, quite like pernicious anæmia (*q. v.*). The patient becomes excessively pale and feeble, the blood shows marked oligocythæmia and poikilocytosis; there are anæmic cardiac murmurs and similar symptoms. This condition also is probably referable to poisonous matter produced by the bothriocephalus latus, absorbed by the intestines, and fatal to the red blood corpuscles. If the tapeworm is promptly expelled there is rapid and complete recovery.

Although many of the symptoms mentioned may arouse suspicion as to the presence of a tapeworm, the *diagnosis* can be made only by finding the joints or eggs of the tapeworm in the dejections. In many cases the patient himself brings some of the segments found by him in the dejections to the physician, but in judging of them a certain caution is always necessary, since shreds of mucus, remains of food, etc., are quite frequently presented to the physician, under the idea that they are segments of tapeworm. But, on the other hand, it is also very important that the physician himself should not forget the possibility of a tapeworm and should himself direct attention in suspicious cases

to the dejections of the patient. If there is a tænia mediocanellata or a bothriocephalus latus, it is usually easy to find the eggs in the feces, but in the case of tænia solium this is more difficult. With tænia solium we usually have to search for segments of the tapeworm in the stools. Many patients with all sorts of indefinite, mild, gastric and intestinal symptoms, or such general symptoms as headache or languor, or even graver disturbances, such as apparent pernicious anæmia, are finally cured when the true cause of the disease is found to be a tapeworm.

If we obtain a few joints of the tapeworm we should endeavor to determine from them the species. If we spread out the pieces of tapeworm between two microscopic slides, the thicker, fatter segments of the tænia mediocanellata, with its many-branched uterus, may usually be distinguished without difficulty from the more tender and more translucent segments of the tænia solium, with a smaller number of lateral branches to its sexual apparatus. The statement of many patients that single segments of tapeworm come from them at other times than when at stool, and that they find them on their underclothing, almost always points to the presence of a tænia mediocanellata in the intestine. The eggs of tænia solium and tænia saginata are, as has been already stated, very similar to each other, while it is easy to diagnosticate the bothriocephalus latus by its eggs, as they are so characteristic (*vide supra*, Fig. 77).

If we suspect a tapeworm, without having secured the certain evidence of segments or eggs in the dejection, it is a good plan to give the patient a mild cathartic, such as castor oil, or a dose of boiled pumpkin seeds, since after this, if the intestine harbors a tapeworm, single portions of it often come away.

Treatment.—The "tapeworm cures," which are recommended in so great a number that we can by no means mention all of them here, but only the most important and the most serviceable, aim at killing or benumbing the worm, and then at removing it from the intestine *in toto* by cathartics.

We usually begin with a so-called "preparatory treatment." This is to cleanse the intestine, especially the large intestine, from old fecal masses, in order to prepare as free a passage as possible for the worm. For this purpose we give the patient a mild laxative, or, better still, a large enema of cold water. Many physicians recommend that there should first be given some active purge, such as calomel or castor oil, but this is exhausting to the patient, and we believe it is not usually necessary. We also forbid for a day or two the use of vegetables, black bread, etc., and prescribe instead a limited diet of white bread, some meat, milk, and coffee. It is a widespread practice to take during the preparatory treatment certain articles of food to "make the worm ill." Among these a salad of finely chopped and very salt herring with onions and garlic is especially recommended. A similar action is also ascribed to strawberries, cranberries, and bilberries. Hence, on the day, and especially on the afternoon, before treatment, we have the patient take a large amount of the articles of food mentioned, such as herring salad.

On the next morning, after everything has been prepared, after the bowels have moved the night before, etc., the patient takes no breakfast, or only some strong sweet black coffee, and then he takes the special anthelmintic. The number of tænicides recommended is, as we have said, very great. At present the following are most in use: Many experienced physicians and we ourselves now employ almost exclusively the ethereal extract of male fern (oleoresina

aspidii). It is true that the reliability of this drug as dispensed by different apothecaries varies, but in most cases, if we can obtain a good fresh preparation, the result is perfectly satisfactory. It should be said, however, that the drug is not entirely free from danger; in exceptional cases large doses have caused symptoms of poisoning, particularly amaurosis, also jaundice; and even death has occurred. We should, therefore, hold firmly to the rule of never exceeding a dose of 2.5 to 3 drachms (gm. 10 to 12). We have never seen unfortunate results from this amount. The best way of administering the drug is in the gelatin capsules which are for sale, of which each contains 15 to 30 gr. (gm. 1 to 2) of the extract. The so-called "gelodurat capsules" (gelatin capsules hardened in formalin), suggested by the chemist H. Rumpel, which contain gr. xv (gm. 1) of the extract of male fern, and only dissolve in the small intestine, being therefore well tolerated by the stomach, are very practical. As a rule, eight of these capsules are sufficient for a complete cure. We generally refrain from administering any further cathartic (castor oil). It is much better to wait quietly and see whether the worm is not passed spontaneously, or, at most, after a few hours to give a careful rectal irrigation, which generally brings out the complete worm (i. e., with the head).

The other tapeworm remedies which were formerly used are at present almost entirely displaced by the extract of male fern. We will mention also the bark of the pomegranate root (cortex radicis punicæ granati), which is given after maceration of 120 to 150 parts in 1,000 of water, subsequently reduced to 150 parts by boiling), the very efficacious pelletierinum tannicum (in doses of gr. vijss. to gr. xxij = gm. 0.5 to 1.5) obtained from pomegranate root, the flores koso (3 to 4 powders of gr. lxxv = gm. 5 in white wine), kamala, etc. Pumpkin seeds (semina cucurbitæ maximæ), which, as has been previously mentioned, are much in vogue as a popular remedy, have been repeatedly recommended by physicians, particularly as they do not share the bad taste of nearly all the other tapeworm remedies. About 120 seeds, or for children half as many, are peeled, pounded up, and mixed with sugar, or else made into an emulsion, and administered.

The treatment is to be regarded as absolutely successful only when we find the head of the tapeworm, as well as its segments, in the patient's dejections. We may best search for the head in the feces by diluting the dejection repeatedly with water, and pouring off the water. The tapeworm then remains at the bottom of the vessel. As it may be that the head has been dislodged even if it has not been found, we should not repeat the treatment for tapeworm until, some months later, there are indubitable signs that the tapeworm still exists.

Every tapeworm treatment is rather drastic, and hence it is well, after the treatment is over, to recommend the patient to be prudent in his diet, and to be careful about his digestive tract for some time. In persons who are very weak, or who have some other disease, we do not willingly undertake to remove a tapeworm without urgent reasons; but in people who are otherwise healthy it is always well to get rid of a tapeworm, even if it causes no severe symptoms. The chief reason for this is that tænia solium might occasion cysticerci in the brain. The best time for undertaking a treatment is when segments or large pieces of the worm come away quite frequently of their own accord. We should never prescribe a treatment on the mere statements or suspicions of the patient.

We must always convince ourselves with complete certainty of the presence of a tapeworm in the intestine.

We must finally mention that the only efficient prophylaxis against acquiring a tapeworm lies in entirely avoiding the use of raw or half-cooked beef or pork. The more widely spread the taking of raw meat is, as in Abyssinia, the more common are tapeworms in man. Certain callings, like those of the cook or the butcher, are also especially exposed to infection.

2. ROUNDWORMS

(*Ascaris lumbricoides*)

Natural History.—Ascarides are pale reddish, cylindrical worms, pointed at both ends, with the sexes in different individuals. The females are 12 to 16 in. [30 or 40 cm.] long, the males about 25. At the cephalic end of the worm are found three lips furnished with fine teeth. The tail is straight in the females and curved in the males. In the female sexual organs (Fig. 83) 60,000,000 of eggs may develop, at a rough estimate. These eggs are often found in the feces of persons who have roundworms in their intestines (see Fig. 77, 8). They have a great capacity of resisting external influences, and a worm-like embryo develops in them in about nine weeks. The further dissemination of roundworms takes place without any intermediate host in this manner: the eggs containing the embryo worms are, through some chance, swallowed and then grow in the intestines into sexually mature worms. Experimental infection with the eggs of roundworms gives distinctly positive results (Lutz, Epstein).

The roundworms inhabit chiefly the small intestine. In severe vomiting they often reach the stomach and are vomited up. In individual cases they have been found in the bile ducts, in the air-passages, and, after perforation of the intestine, in the abdominal cavity. The number of roundworms existing at the same time in the intestine may be very considerable. We find them most commonly in children and in adults from the lower classes. Roundworms have been repeatedly observed to crawl out of the anus, the mouth, or the nose of children during sleep.

The roundworm is also common in hogs and cattle as well as in man.

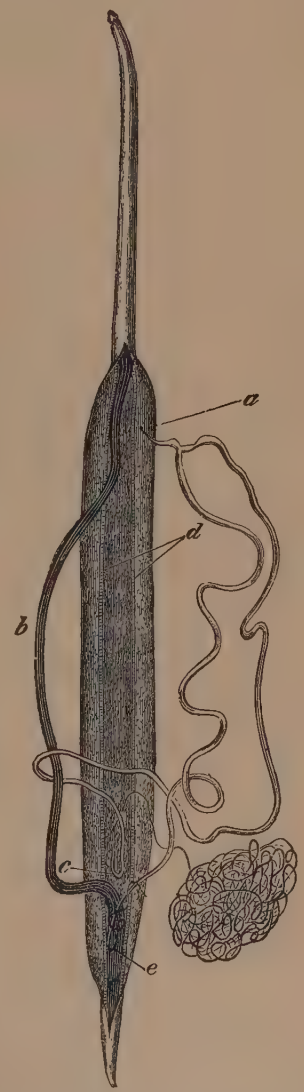


Fig. 83.—(From Heller.) Ascaris lumbricoides. Female, 6 in. [143 mm.] long. *a*. Vagina. *b*. Intestine. *c*. Boundary between the uterus and oviducts. *d*. Longitudinal bands. *e*. Coil of oviducts and ovaries.

Symptoms.—In general, roundworms are innocent parasites, which may exist in large numbers in the intestines without any bad results. In other cases they cause symptoms similar to those ascribed to tæniæ—abdominal pain, languor, itching of the nose and skin, urticaria, burning in the eyes, salivation, etc. —symptoms which are all ambiguous, and whose definite connection with the presence of roundworms it is hard to make out. Occasionally they excite long-continued and persistent vomiting, or violent diarrhea, these symptoms persisting until the worms are expelled, when they promptly cease. Leichtenstern found that it was not very exceptional for children to be noticeably anæmic when harboring ascarides, and to improve remarkably when the worms were expelled.

The cases recorded in literature are quite numerous in which severe nervous symptoms have been caused by roundworms and have disappeared after the removal of the parasites. However cautious we may be in accepting such statements, nevertheless their credibility cannot be wholly denied. We would mention especially convulsions, epileptiform seizures, choreic and cataleptic conditions, contractures, and temporary mental disturbances, which are claimed to be excited by ascarides. It is said that children with ascarides not infrequently exhibit mild nervous disturbances, such as headache, vertigo, mydriasis, and chills. All these symptoms, and in part also those above enumerated, are perhaps not so immediately dependent upon the presence of the worms in the intestinal canal, but are due rather to toxins developed under certain circumstances.

In some cases the presence of ascarides may excite much more severe symptoms by unfortunate accidents, as, for example, sudden suffocation from the entrance of a roundworm into the larynx. When a very large number of roundworms have been present in the intestine, severe symptoms of intestinal stenosis have been observed from their rolling together into a ball. If a roundworm crawls into the bile ducts, it may give rise to jaundice, and even to the development of an abscess of the liver. In the abscesses of the anterior abdominal wall, usually termed "worm abscesses," the roundworms probably play a purely accidental part. We have to do in such cases with perityphlitic abscesses or with inflamed herniæ, which have perforated externally, by which the roundworms which are accidentally found in the intestines pass out, without having any causal relation to the abscess.

Diagnosis.—The diagnosis of roundworms is not usually difficult. Often a few worms appear of themselves, or as the result of a simple laxative. If not, search must be made in the dejections for the eggs of the ascarides. These are always easily found. It is advisable to obtain a particle of the contents of the rectum for the purpose of microscopic examination by introducing an elastic catheter.

Treatment.—The oldest remedy for ascarides is wormseed—santonica. This is best given in the form of an electuary—santonica, 1 drachm (gm. 5); jalap, 15 gr. (gm. 1); and sirup, 1 ounce (gm. 30), to be taken in three doses —in combination with a cathartic. Of late, wormseed, on account of its bad taste, has been almost wholly replaced by santonin, which is derived from it. This is prescribed in 1- or 2-gr. (gm. 0.05 to 0.10) powders, or still more frequently in the form of santonin troches ("worm tablets"), which may be had of any apothecary. It is well to give santonin also in connection with a cathartic, such as calomel. We give the patient one or two doses of santonin in the morning for three days, and on the fourth we give a cathartic. Severe symp-

toms of poisoning (vomiting, dilated pupils, apathy, convulsions, psychical disturbances) have been seen only occasionally from the careless use of it. Milder symptoms, such as a yellowness of the urine and conjunctivæ, and xanthopsia, or seeing everything yellow, are somewhat more frequent.

In exceptional cases santonin fails to expel the ascarides. We may then try male fern or thymol (gr. viij to xxiv, gm. 0.5 to 1.50) in two or three doses given in the course of the day.

3. OXYURIS VERMICULARIS

(Seatworms. Pinworms)

Natural History.—The oxyures are little roundworms, the females $\frac{2}{5}$ to $\frac{1}{2}$ in. [10 or 12 mm.] long, the males only 3 or 4 (see Figs. 84 and 85). The eggs, when they reach the human stomach, develop very rapidly. The embryos, set free, collect in the small intestine and later mainly in the cæcum. As soon as they become sexually mature they pair, and then wander gradually down into the rectum, where they collect in great numbers. When the eggs inside the female become ripe, the female leaves the rectum and lays its eggs outside, where they are soon destroyed (Leichtenstern). The eggs may easily, however, be conveyed by fingers or by articles of diet, or by similar means, to the stomach of the same host or directly, without intermediate host, to some other person, whereupon the development of the worm begins afresh. As will be readily understood, children, with their untidy habits, are much more exposed to infection with the oxyuris than adults. The fact that the female oxyuris never lays its eggs in the intestine explains why the eggs of the oxyuris are rarely if ever found in the stools. The female crawls out of the anus to deposit its eggs. Male as well as female worms are discharged with the stools. The entire cycle of development of the oxyuris occupies about fourteen days. The number of these worms which may be present at the same time in the intestine is very considerable, so that "the entire mucous membrane of the colon is covered with them like a fur."

Fig. 84.

Fig. 85.

Fig. 84.—Oxyuris vermicularis. Natural size. 1. Female. 2. Two males.

Fig. 85.—Oxyuris vermicularis, enlarged. *a*. Mature female, not yet impregnated. *b*. Male. *c*. Female containing eggs.

Symptoms and Treatment.—The oxyures found in the upper portions of the intestine and in the cæcum cause no symptoms whatever, but in the lower part of the rectum their presence causes local symptoms, especially a very

severe feeling of itching and burning in the anus, which makes the child constantly scratch and dig with his fingers. This itching of the anus is most severe at night in bed. In girls the oxyures frequently travel into the vagina, by which an intense itching is also set up there, which sometimes leads to masturbation. In some cases in boys and men, oxyures have been found to be the cause of abnormal sexual irritation. Other clinical symptoms are rare, but sometimes there may be anæmia or nervous disturbances, and in rare cases marked intestinal catarrh, or inflammation of the vulva.

The diagnosis of oxyures is not difficult. Our attention is called to the itching of the anus, etc., and we look for worms. Single worms are easily found in the dejections, and often on the skin about the anus.

Treatment is directed first to the expulsion of the threadworms from the rectum, and the prevention of any fresh self-infection. For this purpose the chief means are large injections, which must be employed persistently, once or twice a day, for weeks; for fluid we may choose simple warm water, a one-per-cent solution of tannic acid, or water containing vinegar, salt, or glycerin. Injections of 1 or 1.25 drachms (gm. 4 to 5) [for children gr. xxx to xl = gm. 2 to 2.5]) of guiasanol (a guaiacol preparation made by the Höchster dye works) are claimed to be particularly efficacious. Injections containing corrosive sublimate or carbolic acid should never be employed because of the danger of poisoning. It is difficult to remove completely the oxyuris from the upper part of the intestine, particularly from the cæcum. For this purpose the most efficient remedy seems to be santonin, perhaps combined with calomel or castor oil. The itching at the anus is alleviated by cold compresses or by the inunction of small amounts of unguentum hydrargyri.

Extreme cleanliness is very important. The anus should be frequently washed, and also the fingers, particularly the finger nails, to prevent the spread of the parasites.

4. ANCHYLOSTOMA DUODENALE

(*Dochmius seu Strongylus duodenalis. Uncinariasis. Hookworm Disease*)

The *anchylostoma duodenale* is a worm first observed in upper Italy and in Egypt in large numbers, of which many inhabit the upper portion of the small intestine, especially the duodenum, but also the jejunum and ileum. The male is $\frac{1}{4}$ to $\frac{2}{5}$ in. [6 to 10 mm.] long, the female 10 to 18. At the cephalic end (Figs. 86 and 87) is found a bell-shaped mouth capsule, which is provided with two small teeth on its dor-

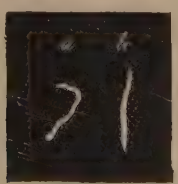


Fig. 86.—Anchylostoma duodenale, natural size. *a*, Male. *b*, Female.

Fig. 87. — (From Heller.) Anchylostoma duodenale, enlarged. Head with bell-like mouth.

sal edge, and four larger curved teeth on its ventral edge. With this sucking and biting apparatus the worm fixes itself firmly, like a wet cup, on the intestinal mucous membrane, and is nourished by the blood which it sucks out. The place in the intestine to which an anchylostoma has fastened may be recognized in the cadaver as a little ecchymosis. The worms sometimes bore completely into the inner part of the mucous coat.

The eggs of the anchylostoma are discharged in great numbers with the feces (*vide infra*). From these the larvæ develop and thrive, particularly in the dirty water of mud puddles and ditches; and by means of water that is spattered, or more often by dirty hands, they reach the mouth and the intestinal canal of man again, and then rapidly develop to sexual maturity. Recently several investigators (Loos, Schaudinn, and others) have shown experimentally that the anchylostoma larvæ can also enter the body through the skin and reach the intestinal tract (presumably through the lung capillaries into the bronchial tubes and from there the œsophagus and stomach). Probably this method of infection actually plays a great rôle.

If the intestine harbors many anchylostomata, the small but constant loss of blood caused by them is not without influence on the organism. In addition, there are probably specific toxic influences which have an injurious effect upon the red blood corpuscles. The symptoms of a severe anæmia gradually develop. Griesinger first made the discovery, in the year 1854, that the disease long known by the name of "Egyptian chlorosis" was caused by the anchylostoma duodenale. Since then confirmatory observations have been made in many parts of the tropics.

The disease caused by the anchylostoma has become particularly known in Europe because of the great frequency with which it occurred among the Italian laborers who were employed in building the Saint Gothard Tunnel. Since then many well-established cases have been found in other lands; e. g., Hungary; Germany, particularly in the brickmakers of the Rhine; also repeatedly in miners and laborers in tunnels.

The symptoms of the disease consist, as we have said, of a gradually increasing general anæmia, for which no special organic lesion can be made out objectively as a cause. The patient also suffers from very great general weakness and languor, dyspnœa, palpitation, headache, œdema, etc. The changes in the blood (oligocythæmia, poikilocytosis) are precisely similar to those seen in pernicious anæmia. The disease may last for months, or even years, and it often ends fatally, if it be not recognized and treated in time.

Leichtenstern has made numerous and accurate observations with regard to the brickmakers of Cologne. He states that no symptoms are observed for three or four weeks after infection with the embryos of the anchylostoma. Some five or six weeks after infection, when the parasites become sexually mature and breed, there appear bloody diarrhea, intestinal colic, and accompanying progressive anæmia. At this time there is probably more shifting about of the parasites in the intestine, while they later become more fixed. This explains why the disease evinces a more acute and severe character at first, and then takes on the form of a chronic anæmia, with great diminution or cessation of the bloody stools.

The diagnosis is easy if we only think of the possibility of anchylostoma. It is not very difficult to find an abundance of eggs in the feces. They are of

oval shape and characterized by the frequent presence in them of two or more spherules due to subdivision (see Fig. 88). The worms themselves are not usually found in the stools unless anthelmintics have been administered. Charcot's crystals are often found in the dejections previous to the appearance of the anchylostoma eggs (Leichtenstern). There is always an increase of the eosinophile leucocytes in the blood.

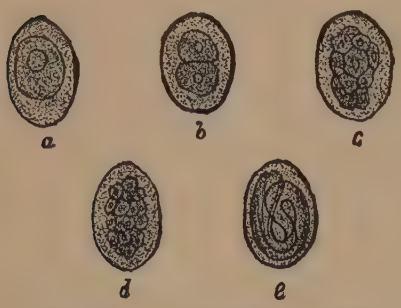


Fig. 88.—Egg of anchylostoma duodenale. *a* to *d*, various stages of segmentation; *e*, with embryo. (From Perroncito and Schulthess.)

If the disease is recognized, treatment is usually satisfactory. We prescribe the same anthelmintics as for the other intestinal parasites, particularly extract of male fern in suitable doses (2 to 3 drachms [gm. 10 to 12]), besides laxatives and injections. In this way it is often possible to banish the parasites entirely from the intestinal canal, and thus to induce, even in severe cases, a complete and often surprisingly rapid recovery. Besides extract of male fern, thymol has been found an effectual remedy. Of this, about 2.5 to 3.5 drachms (gm. 10 to 14) are given daily in doses of 30 gr. (gm. 2) each. For children the dose is proportionately less. Leichtenstern recommends that a few doses of calomel and one or two injections should be administered on the day preceding the specific treatment with male fern or thymol.

The prophylaxis from infection with anchylostoma is an important matter in industrial hygiene. The points chiefly to be considered are to enjoin the greatest cleanliness upon the workmen, to arrange properly for the reception and removal of the excreta, and to provide good drinking water. [The type seen in the United States is the *Uncinaria americæ* (or *Necator americanus*), and has been shown by Stiles to be prevalent and important in the South. Besides the anæmia, eosinophilia is an important sign. Thymol is the favorite remedy—30 gr., repeated in two hours, and followed two hours later by a purge, preferably not castor oil, as this may dissolve the thymol and cause toxic symptoms.]

5. TRICHOCEPHALUS DISPAR

(*Whipworm*)

The *trichocephalus dispar* is a worm 1.3 to 2 in. [4 or 5 cm.] long, whose anterior part is very thin, but whose posterior part is decidedly thick (Fig. 89). It resides chiefly in the cæcum, but also in the colon, where it is often found in small numbers, and sometimes in abundance. The yellow or reddish-brown eggs of the parasite are characteristic, and they may be easily found in the feces upon microscopic examination, but the parasites themselves scarcely ever come away spontaneously with the dejections. As a rule, the trichocephalus has no clinical significance, but lately Moss-

Fig. 89.—(From Heller.) Trichocephalus dispar.

brugger has reported a few cases of severe trouble in children, due to this parasite. The infection was supposed to be due to eating earth. The symptoms consisted in great anæmia and in long-continued and violent diarrhea, with watery movements containing mucus and blood. The dejections also showed many eggs of the trichocephalus and Charcot's crystals. Treatment was very difficult and tedious, for the ordinary anthelmintics seemed to have slight effect. The most promising remedies in such cases would probably be extract of male fern and thymol.

SECTION VI

Diseases of the Peritoneum

CHAPTER I

ACUTE PERITONITIS

(Inflammation of the Peritoneum)

Ætiology.—There are two ways by which inflammatory agents most frequently reach the peritoneum: one is from the gastro-intestinal tract, and the other—in women—is from the genitals.

All the diverse forms of ulceration which attack the digestive canal may involve the serous layer. In such a case an inflammation arises which is at first limited, but which may under certain circumstances become more extensive. This inflammation may be regarded as analogous to that of the pleura in pneumonia; but the anatomy of the stomach and intestine is such that very often an ulcer in their walls ends in a complete perforation. If this occurs, the inflammatory germs contained in the primæ viæ at once escape into the peritoneal cavity and there excite an inflammation, which, from the specific character of its cause, is invariably purulent, and very frequently is at the same time septic or ichorous. The possibility of a peritonitis due to perforation, as a result of the various ulcerative processes of the stomach and the intestines, has been frequently referred to in the previous sections of this work. Thus it may occur in simple ulcer and in ulcerating cancer of the stomach; in typhoid, tubercular, or dysenteric ulceration of the intestine; in ulceration of the intestine above intestinal stenoses of many varieties; and in the small ulcers of the vermiform appendix due to the pressure of hard substances.

The female organs of generation are the other important source of peritonitis. In labor and premature delivery the genital tract was formerly often directly infected. At the present time this complication has fortunately become infrequent, thanks to the advances in asepsis. The various forms of inflammation which are thus set up, including endometritis, metritis, and parametritis, may in several different ways reach the peritoneum. A septic inflammation of the endometrium may involve the peritoneum by direct extension up the Fallopian tubes. In other cases it is through the lymph vessels

that a purulent metritis or parametritis spreads to the peritoneum. The larger parametritic abscesses may break into the peritoneal cavity. It is to be particularly noticed, however, that in many cases of septic puerperal peritonitis the uterus and its appendages are in a perfectly normal condition, having served merely as a gateway to the inflammatory agents without suffering any harm themselves.

Besides these two chief sources of peritonitis, numerous others are possible, although much less frequent.

Sometimes peritonitis is due to an extension of inflammation from other abdominal viscera. Hepatic abscess, suppurating hydatid cysts of the liver, ulcer of the biliary ducts, splenic abscess or infarction, purulent nephritis or pyelitis, abscess near the bladder or in the prostate, suppurating ovarian cysts, tubal pregnancy, psoas abscess, and Pott's disease—all these may produce peritonitis, either by direct extension or by perforation.

It is worthy of note that peritonitis may occur as a sequel of pleurisy. The pleural and peritoneal cavities are directly connected by the lymph-vessels of the diaphragm; and empyema, as well as tuberculous pleurisy (see next chapter), may spread to the peritoneum.

Penetrating wounds of the abdomen are a fruitful source of acute peritonitis. Surgical operations upon abdominal organs come under the same head. A large number of laparotomies proved fatal before antisepsis was introduced, because the inflammatory germs thus admitted excited a diffuse septic peritonitis. Even tapping the abdomen for ascites may cause acute peritonitis if the trocar is not aseptic. Abdominal injuries, in which the walls are not penetrated, very rarely, if ever, give rise to peritonitis. One way in which they have been said to produce it is by exciting internal hemorrhage. In the newborn, peritonitis exceptionally results from infection through the navel.

Far less frequent are those cases of acute peritonitis which occur as a part of certain general diseases. In this class belong the suppurative peritonitis sometimes developing by a hematogenous route in general sepsis and the peritonitis of acute articular rheumatism (*q. v.*). The latter is rare and usually benign. It must be regarded as analogous to the "rheumatic" inflammation which occurs in other serous membranes, including the endocardium, pericardium, and pleura. It is possible that rheumatic peritonitis of this sort may exceptionally occur as an apparently primary disease. It is a well-established fact that peritonitis may also develop in the course of acute or chronic nephritis. This we have ourselves observed. It is, of course, comparable with the inflammation which not infrequently attacks the pericardium and pleura in the course of nephritis, which probably depends upon the retention in the blood of the solids which ought to be excreted with the urine. We must also mention that in very rare instances a specific gonorrheal peritonitis occurs, either in connection with a constitutional infection from gonorrhea, perhaps associated with gonorrheal synovitis or endocarditis, or from the direct extension of gonorrheal inflammation from other parts to the peritoneum.

From all that has been said, it is evident that, from a purely ætiological standpoint, peritonitis is by no means a stereotyped and uniform disease. The actual pathogenic agents, exclusive of the rare cases of mere intoxication, are always bacteria, but bacteria of diverse kinds. In the cases of peritonitis

due to perforation, the bacterium coli seems to play an important part, and often also streptococci are influential. The cases of septic or puerperal peritonitis are mostly occasioned by streptococci; rarely the peritonitic exudation has been found to contain pneumococci, staphylococci, gonococci, proteus forms, and other germs. Not infrequently there seems to be a mixed infection from the start.

Pathology.—Like the analogous inflammations of the pleura and pericardium, peritonitis is divided into different varieties, according to the character of the inflammatory exudation. The nature of the exciting cause of most cases of peritonitis is such that by far the most frequent variety is the fibrinopurulent. If the process involves the entire peritoneum—that is, if there is a "diffuse general peritonitis"—we generally find upon opening the abdomen that the parietal layer of the peritoneum and the outer surface of the intestinal coils are distinctly reddened from marked vascular injection. There may even be small ecchymoses here and there. The serous membrane is clouded, a result partly of desquamation of its endothelium, and partly of the more or less abundant fibrinous exudation which covers the peritoneum with a sheet of coagulated fibrin. Very often the coils of intestine have formed numerous adhesions with one another (compare pleuritic adhesions). In cases of brief duration these can still be easily broken up, but after a prolonged illness they are extremely firm. There is usually also some free, fluid, fibrinopurulent exudation in the abdominal cavity. Its amount varies greatly. Sometimes there is only a small quantity of opaque fluid in the dependent portions of the cavity; sometimes there are many quarts, causing great distention of the abdomen. The exudation seldom inclines to a seropurulent character. It is usually predominantly purulent. Very often the purulent exudation undergoes decomposition into the offensive sanious fluid of septic peritonitis. This is particularly apt to occur when the disease originates from an intestinal perforation or from puerperal poisoning. The perforation through the walls of the intestine is sometimes so large as to admit considerable amounts of intestinal gases and feces into the peritoneal cavity. It is also possible that the putrefaction of peritoneal exudations may generate offensive gases. In rare instances the exudation is hemorrhagic; but most cases of hemorrhagic peritonitis do not belong here, but come rather under the tubercular or cancerous forms (*vide infra*).

In severe and protracted cases of peritonitis the intestine is involved to a certain extent. There is a collateral inflammatory œdema of its walls, causing a considerable increase in thickness, while at the same time they are often friable and easily torn. The weakness of the muscular layer of the intestine may amount to complete paralysis, and thus permit excessive intestinal tympanites, either diffuse or local.

Milder forms of general peritonitis with serofibrinous, or chiefly serous, exudation are relatively infrequent. Under this head would come certain apparently primary and usually chronic cases with favorable issue, and also the peritonitis which sometimes occurs as a sequel of an ascites which has existed for some time (see next chapter). Probably also in those rare cases of peritonitis arising in the course of acute rheumatism and ending in recovery, the exudation has been serofibrinous.

We have spoken thus far of diffuse general peritonitis, but cases are not

rarely seen of circumscribed or "encapsulated" peritonitis. Here, also, we have mild varieties with fibrinous exudations on the one hand, and on the other purulent inflammation. The milder inflammation is a result of the extension of the most varied forms of inflammation from neighboring organs. Thus, deep intestinal ulcers, for example, give rise to a mild circumscribed inflammation of the corresponding portion of the serous layer. A similar condition results from superficial splenic infarctions; from various hepatic diseases, when they reach the surface of the liver, and from numerous pathological conditions of the female genitals. In many of these cases the peritonitis takes a chronic course and leads to adhesions, and hence is called adhesive peritonitis.

Circumscribed purulent peritonitis has precisely the same ætiology as the general form, with this single difference, that firm adhesions are quickly formed around the spot whence the inflammation proceeds, limiting it and preventing it from involving the entire peritoneum. It occurs most frequently as a purulent perityphlitis (*q. v.*), consequent upon perforation of the vermiform appendix, and also as pelvic peritonitis, which is a possible sequel of most of the forms of puerperal inflammation to which the uterus and its appendages are liable. But we may also have encapsulated purulent peritonitis after the perforation of gastric and intestinal ulcers, or of suppurative processes in the biliary passages, and from similar causes. If the abscess is situated directly below the diaphragm it is termed subphrenic.

Histologically considered, acute peritonitis is perfectly analogous to the inflammatory processes which attack other serous membranes. The endothelium becomes degenerated, and, for the most part, is cast off. There is an exudation from the blood vessels of a fibrinous fluid, which is partly coagulable, and with this exudation round cells escape in greater or less abundance. In the further progress of the disease there is an inflammatory new growth of vascular connective tissue, which probably originates chiefly from the endothelium and the permanent tissue cells, but, according to some, starts in part also from the wandering cells. The new formation of blood vessels certainly seems to be due chiefly to budding from the capillaries of the serosa. Thus arise the adhesions of connective tissue and the false membranes found in chronic cases between the different coils of intestine. They lead in process of time to marked thickening and retraction of the omentum and mesentery (*peritonitis deformans*). Most cases of purulent peritonitis prove fatal in the early acute stage. If a case recovers, the exudation undergoes fatty degeneration, and its cellular constituents are thus disintegrated and then are absorbed.

The results of circumscribed purulent peritonitis are detailed in connection with the clinical history.

Clinical History.—1. *Acute General Peritonitis.*—The following description applies chiefly to the severe purulent form, the one by far most frequently met with. It occurs in most instances after perforation, in puerperal cases, and after external injuries, such as surgical operations. In most of these cases the peritonitis is a secondary disease, so that it must obviously be greatly modified in its general characteristics and behavior by the original trouble. In the first place, the onset is modified. Many cases of peritonitis due to perforation begin almost abruptly, the patient having been previously in perfect health. Thus, as already mentioned, the first indication of a gastric or

duodenal ulcer may be given by perforation. Some cases of perforation of the vermiform appendix present equally sudden and unexpected symptoms.

There are many other cases in which the symptoms of peritonitis supervene upon those of some grave disease already existing. For example, typhoid fever, intestinal tuberculosis or intestinal stenosis, may, by causing perforation, excite a peritonitis. Here the symptoms of this secondary disease may be more or less completely veiled by the other grave local and constitutional disturbances.

Again, an acute general peritonitis may, as we have already said, be the sequel to a local and circumscribed inflammation of the peritoneum. Thus, a purulent perityphlitis, or a purulent puerperal pelvic peritonitis, may finally become universal. In such unfortunate cases the change in symptoms is often gradual, and is not clearly pronounced.

We have now indicated certain variations from the general course of the disease; but, nevertheless, almost every case of acute general peritonitis, whatever its ætiology, presents clinical symptoms which are so characteristic and typical that a general description of the disease will be both easy and advantageous.

The symptoms of acute peritonitis form two groups, the local and the constitutional. The latter are the result of the local disturbance acting upon the general condition of the patient.

Of the local symptoms, pain deserves to be named first. It is usually the earliest symptom, and, as the disease progresses, it is generally the excruciating abdominal pain which attracts most attention. The localization of the pain in the beginning of the illness may be of diagnostic value in doubtful cases, if such as to indicate the possible starting point of the inflammation; for example, the vermiform appendix or a gastric ulcer. Later, the pain extends over the whole abdomen. As a rule, there are brief remissions followed by fresh exacerbations. The pain is aggravated by voluntary movements, by deep inspirations, and probably by intestinal peristalsis. The abdominal tenderness is often extreme in peritonitis, and is very characteristic. The gentlest palpation is torture, and often the slightest pressure of the bedclothes is almost unbearable. Frequently the greatest tenderness is in the umbilical region.

Acute peritonitis seldom exists without pain. The exceptions to this rule are seen chiefly in patients who are extremely prostrated, and whose sensibility and intelligence are much impaired. Here the peritonitis may escape notice—as in severe typhoid or in the last stage of tuberculosis. Comparatively slight pain is supposed to occur in those cases in which the parietal peritoneum is not markedly involved in the inflammation.

On physical examination, the abdomen, as a rule, is found to be distended. This is an early symptom, and gradually becomes more and more pronounced. It is due mainly to the intestinal tympanites which we have already mentioned, which sometimes becomes very great if the muscular fibers of the intestine are paralyzed. In the later stages the liquid effusion into the peritoneal cavity of course contributes to the prominence of the abdomen, but even then the distention is seldom so uniform or so broad as in ascites. In peritonitis, coils of distended intestine can often be recognized by their characteristic contour through the abdominal wall. On palpation usually the most striking fact is the general increased rigidity of the abdominal walls dependent

upon a reflexly increased tonicity of the abdominal muscles. This increased resistance, the "*défense musculaire*" of the French, is explained as being a protection against painful pressure. It also accounts for the frequent disappearance of the abdominal reflexes.

Palpation also often shows a very characteristic diversity in the sense of resistance in different portions of the abdomen, occasioned by variations in the amount of exudation, by adhesions, or by the dilatation of some of the intestinal coils, and similar causes.

In general, if the abdominal wall is yielding and thin, the peritonitic distention will be greater, so that it is most marked in puerperal cases, where the preceding pregnancy has rendered the walls lax. In a person with powerful muscles and tense abdominal walls the convexity of the abdomen is seldom great. In some cases there is no convexity whatever. The walls may be as hard as a board, and the abdomen flat or slightly concave. In such cases the diagnosis may be difficult. Sometimes, again, the original retraction of the abdominal walls is succeeded by more or less distention of the abdomen.

Percussion over the distended intestinal coils yields a resonant and usually tympanitic sound. It is not till a considerable amount of liquid effusion has collected that there is dullness, most marked in the dependent portions of the abdomen. If there is much tympanites, however, quite a large effusion may exist without being detected on percussion. Percussion also gives results analogous to those of palpation, in that there is often a diversity in the quality of the resonance of different portions of the abdomen in peritonitis.

Usually there is too much pain to permit a careful examination of the change of dullness consequent upon change of decubitus. In general the numerous adhesions between the separate coils of intestine also interfere with the free motion of the peritonitic exudations.

Percussion not only gives information about the existence of a liquid, purulent effusion, but is also of value in determining the level of the diaphragm, as affected by abnormal abdominal distention. The upper limit of hepatic dullness is raised to the fifth or even the fourth rib. The heart is also pushed up. There is a tympanitic resonance above the margin of the ribs on the right side.

The diaphragmatic movements in respiration are very slight, partly on account of the increased intra-abdominal pressure, and partly on account of the sensitiveness of the abdomen. For this reason peritonitis patients almost always show a pronounced costal type of respiration.

The area of hepatic dullness is not only displaced upward, but is also evidently diminished. This is due in part to coils of distended intestine overlapping the anterior edge of the liver, and in part to the organ being tilted upward in such a way that its area of contact with the anterior wall of the body is less than normal. Formerly the total disappearance of hepatic dullness was regarded as a sure sign that gas has escaped from the intestine into the abdominal cavity. The inference is not always correct. The liver may be displaced backward by coils of intestine, and hepatic dullness be thus abolished, although there is no air free in the peritoneal cavity.

If there is a considerable effusion, it is possible, as in ascites (*q. v.*), to get a sensation of fluctuation by gentle, quick palpation. If, after the physical examination, the existence of a peritonitic exudate is still doubtful, posi-

tive evidence is furnished by an exploratory puncture which, at the same time, permits us definitely to determine the character of the exudate.

As a rule, auscultation of the abdomen does not throw much light on a case of peritonitis. In the distended coils of intestine, not infrequently at the beginning of the disease, we hear all sorts of gurgling sounds. In the later stages the intestinal paralysis which is present causes a cessation of all peristalsis. Sometimes we hear a peritonitic friction sound, due to the movements of respiration, causing two rough surfaces to rub against each other. In particular, perihepatic friction is heard not very infrequently.

If the results of physical examination leave us still in doubt as to the presence of a peritonitic exudation, we may attain certainty by an exploratory puncture, and at the same time, if there be any exudation, determine its character.

The digestive apparatus is almost always disturbed by any severe case of peritonitis. In all severe cases the tongue is dry, fissured, and often thickly coated. A moist tongue is regarded prognostically as a good sign. The appetite disappears entirely. On the other hand, there is usually a torturing thirst.

As to the stomach, vomiting is the most important symptom. Vomiting is often seen early in the disease, and recurs frequently as the illness progresses. It sometimes is spontaneous, and sometimes follows the ingestion of food. If spontaneous, the vomitus consists of watery mucus, usually of a greenish tinge. We do not know absolutely the cause of the vomiting in peritonitis. Apparently it is in part a reflex action, excited by the inflammation of the serous membrane. Possibly the external pressure of the exudation also affects the stomach, and it may be that absorbed toxins also excite vomiting. It must be added that vomiting may be absent in acute peritonitis. This is seen when the patient is comatose, and sometimes also when the peritonitis has developed upon perforation of a gastric ulcer, because the contents of the stomach are thus emptied out through the hole in its walls. The vomiting is usually accompanied by frequent eructations.

Of the intestinal symptoms, the reader has already become acquainted with the tympanites, and also with the fact that it is due mainly to a paresis of the muscular fibers of the intestine. This same muscular weakness furnishes an obvious reason for the persistent constipation usually observed in peritonitis. If the intestinal paralysis progresses, so-called paralytic ileus (*vide supra*) may finally develop. On the other hand, we may have diarrhea instead, from increased peristalsis and secondary intestinal catarrh. Especially in some forms of septic puerperal peritonitis, diarrhea is frequently observed. Micturition is usually scant and often painful. This is probably dependent upon an involvement of the peritoneal covering of the bladder. Not infrequently the urine contains some albumen, and occasionally also indican.

The pushing up of the diaphragm has a noteworthy effect upon the thoracic organs. The lower lobes of the lungs are compressed, so that considerable dyspnœa results. The heart is likewise crowded upward, so that the apex beat is usually to be felt in the fourth intercostal space.

Every case of acute peritonitis that is at all extensive has marked constitutional effects. These are in part the result of the wakefulness due to pain, and the restlessness and fever. But perhaps there are also reflex inhibitory

influences, originating in the irritation of the peritoneal nerves and affecting chiefly the heart, just as Goltz in his well-known experiment killed a frog by blows upon the abdomen. The chief factor, however, is in all probability the toxins which are readily absorbed from the peritonitic exudation by the peritoneum, and thus enter the circulation. They produce a vasomotor paralysis of the splanchnic area, so that all the other vascular regions are emptied.

There is no other disease, except internal strangulated hernia—and the effect of that is perfectly analogous—which produces general collapse and weakness so quickly as does peritonitis. The imperfectly nourished and poorly filled heart acts progressively weaker. The countenance (*"facies abdominalis"*) is rapidly altered, the cheeks fall in, and the eyes become hollow. The nose grows sharp and cool, the lips and tongue dry. The skin of the extremities is also cool and bluish, as a result of impaired circulation. The peritonitis has hardly begun before we find the pulse small and soft. In many severe cases the pulse finally becomes almost imperceptible. At the same time the pulse rate increases, as is usual in collapse from any cause, so that 120 to 140 beats per minute is not an exceptional rapidity. Only in exceptional instances is there no increased rapidity of the pulse.

The temperature varies greatly in different cases. It may be high in the rectum, although the skin feels cool. Still, very high fever is not usual; and there are often considerable remissions. We even frequently observe the subnormal temperature of collapse. In general, streptococcic peritonitis runs its course with higher temperature than that caused by the colon bacillus. In the latter, fever may even be almost entirely absent. The number of respirations per minute is usually 30 to 40. This increased rate is due not only to the compression of the lower lobes of the lungs, but also to the pain caused by full inspirations and to the impeded circulation.

The intellect usually remains unimpaired to the end. Not infrequently just before death, a striking euphoria appears. Sometimes there may be mild delirium, or an approach to stupor, toward the close of the disease.

The course of acute general peritonitis in the great majority of cases is unfavorable. With the appearance of the grave symptoms just depicted the prognosis becomes almost hopeless. The course of the disease is also comparatively rapid. Marked variations in the intensity of the symptoms are infrequent. The grave local and constitutional symptoms persist, and, as a rule, the patient dies at the end of a few (two to six) days. Still, it is not well to make general dogmatic statements as to the clinical history, for the ætiology of each individual case impresses upon it individual characteristics. A peritonitis resulting from gastric or intestinal perforation is usually quickly fatal. The same is true of almost all cases of puerperal septic peritonitis. In a few cases, however, the inflammation is limited, by the encapsulation of the exudation. These may finally end in recovery through perforation of the abdominal walls or perforation into the intestinal canal. Now and then an acute general peritonitis may assume a chronic form. The effusion is mostly reabsorbed, and the newly formed adhesions and false membranes contract into firm bands of connective tissue. The liver, spleen, and other abdominal viscera acquire a tough coating of connective tissue. The omentum and mesentery are shortened and thickened. Indeed, the omentum may roll itself almost completely up. Although the clinical symptoms become less severe,

weakness usually persists, with gradual exhaustion and death. Often the intestine is so bent or pinched as to give rise to grave symptoms from stenosis.

Recovery from acute general peritonitis is very exceptional. If seen, it is usually in mild cases, such as sometimes occur after menstruation, abortion, or labor. A favorable outcome is the rule, also, in those very rare cases that develop in the course of an acute articular rheumatism, as well as in certain forms of gonococcus peritonitis, especially those that occur in women subsequent to a gonorrheal endometritis and salpingitis, and also in pneumococcus peritonitis. This last is usually observed in children; it has a sudden onset with simultaneous gastrointestinal symptoms and a tendency to encapsulated abscess formation below the umbilicus. In these groups of cases the inflammation is either of a serofibrinous nature and is finally absorbed, or it is suppurative, and becomes limited early in the disease with the formation of a circumscribed abscess, which ruptures spontaneously either into the intestinal lumen or externally (as through the umbilicus in pneumococcus peritonitis), or else can be opened by operation.

2. *Acute Circumscribed Peritonitis.*—The local symptoms of this are essentially the same as we have just ascribed to the general form; except that, a smaller extent of tissue being involved, they are correspondingly limited. The pain and tenderness are confined mainly to one region, but its boundaries are rarely sharply defined. On palpation of this region, we find an increased resistance which is sometimes almost like that produced by a tumor. If there is an encapsulated effusion, we may detect fluctuation, particularly if the abscess is going to point outward. On percussion over the affected spot, there is either dullness or a muffled tympanitic resonance.

The constitutional symptoms are likewise those of general peritonitis, only usually less severe. Reflex vomiting does occur, but is seldom so persistent as in the diffuse inflammation. The physical weakness and symptoms of collapse are decided, but do not usually become extreme. There is generally an irregular fever, which may now and then assume an intermittent, pyæmic character. Most cases run a chronic course. If the illness be very much prolonged, death may finally ensue from general debility. Recovery is possible if the pus can be let out. This may be accomplished either by the surgeon or by Nature. Spontaneous discharge of the abscess may take place through the abdominal walls, into the intestine, or even, in rare instances, through the pleura into the lungs. But if the pus finds its way into the general peritoneal cavity, the peritonitis becomes diffuse and causes death.

To describe in detail each separate variety of circumscribed peritonitis would occupy too much space, and would also lead to useless repetitions. We have already spoken at some length of one especially important form—namely, perityphlitis. Perimetritis and pelvic peritonitis are chiefly puerperal affections, and are fully discussed by writers on gynæcology.

The so-called subphrenic abscesses deserve a more exhaustive discussion at this point. They develop below the diaphragm, between it and the liver on the right side, and between it and the stomach, colon, and spleen on the left. Right-sided subphrenic abscesses usually originate from suppurative processes in the liver, bile passages, the right kidney, the appendix, etc. Left-sided subphrenic abscesses most frequently develop from perforated gastric ulcers and much more rarely from the left kidney, the spleen, the left lobe of the

liver, etc. In individual cases, caries of the lower ribs and suppurative processes in the lungs and the pleura that extend through the diaphragm, also lead to subphrenic abscesses. If, in a stomach or colon perforation, the abscess contains air, or gases have developed in it as a result of decomposition, this type of abscess is designated as a subphrenic pyopneumothorax. The symptoms of subphrenic abscess are local pain, fever, and, depending upon the position and size of the abscess, dullness and other physical signs. The differentiation from empyema and pneumothorax is not always easy. The most important differential point in favor of subphrenic abscess is the demonstration of the presence of the respiratory mobility of the lower boundary of the lung. Sometimes the irregularity of the upper boundary of dullness is striking, especially in those cases where the abscess develops on the right side, anteriorly, between the liver and diaphragm. If the abscess contains air, metallic auscultatory phenomena are present, as in pneumothorax (coin sound and metallic splashing sounds on succussion). If vesicular breathing can be demonstrated above the tympanitic area, then it is certain that the cavity lies below the diaphragm. Still, it should be noted that pleuritic exudates, at first serous, but later also purulent, frequently develop secondarily to a subphrenic abscess. This and other complications (pericarditis, perforations, etc.) often cause the physical condition to become still more involved and difficult of interpretation. The X-ray examination frequently gives important information. Practically, the chief point is to recognize the presence of pus in time. For this purpose exploratory puncture is of the utmost importance, and must never be neglected in these cases. As soon as suppuration has been demonstrated, proper surgical treatment with sufficiently free opening of the abscess cavity must be instituted. Almost all cases of subphrenic abscess that are not treated surgically run an unfavorable course.

Of the other forms of circumscribed suppuration in the abdominal cavity, perinephritic abscess will be considered more in detail later. I would refer here, briefly, to a form of localized suppurative peritonitis, observed particularly in children, in which a painful fluctuating tumor develops above the left inguinal fold, and which usually ends favorably by perforation of the abscess into the rectum. The infection of the peritoneum in these cases probably comes from the descending colon or the sigmoid flexure.

Diagnosis.—The diagnosis of peritonitis is in many cases an easy matter, when we have the characteristic symptoms of tenderness and tympanites, vomiting, and collapse. Often the starting point of the inflammation is equally obvious in cases of secondary peritonitis supervening upon some disease which we have already clearly recognized, such as typhoid fever, gastric ulcer, or puerperal diseases. But where the peritonitis is apparently primary, we must inquire carefully into the previous history and the earliest symptoms of the attack, in order to form even a surmise as to ætiology.

The diagnosis is sometimes greatly obscured by the fact that under certain circumstances very similar symptoms may be excited by other disorders affecting the intestines. Thus, in typhoid fever there may be great tympanites and grave constitutional symptoms, with abdominal pain, so that peritonitis may be diagnosticated, while the autopsy, if there be one, discloses no signs of it. Deep ulcers of the intestine, however produced, may give rise to such great abdominal tenderness as likewise to simulate peritonitis. The differen-

tial diagnosis between peritonitis due to perforation and acute intestinal obstruction (*q. v.*) is often very difficult. In both, when there is severe constitutional infection, the symptoms are almost the same, and, furthermore, diffuse peritonitis may occasion such a paralysis of the intestine or such excessive tympanites as to prevent any motion of the bowels, and even to cause fecal vomiting (the so-called paralytic ileus). On the other hand, intestinal obstruction is not infrequently complicated by general peritonitis, so that the two conditions may both exist simultaneously. The following are the chief points of distinction between them, although even these are not infallible:

PERITONITIS	INTESTINAL OBSTRUCTION
1. Begins with fever and with abdominal pain, often localized.	1. Begins without fever. Pain absent in obturation (*vide supra*).
2. Abdomen very sensitive to pressure, hard, and tense.	2. Abdomen at first soft and not especially tender upon pressure.
3. Pain tends to abate as the disease goes on.	3. Abdominal pain gradually and constantly increasing.
4. No visible peristalsis of the intestines.	4. Sometimes visible peristalsis; intestinal coils can be felt like rolls.
5. Seldom fecal vomiting, frequently hiccough and simple vomiting.	5. Distinct fecal vomiting, hiccough rare.
6. Exudation demonstrable in the peritoneal cavity.	6. No free exudation in the peritoneal cavity, or only slight, later.
7. Passage of gas not entirely stopped.	7. Complete obstruction.

Another diagnostic error, which we have already mentioned, is not infrequent—that is, the occurrence of peritonitis may be entirely overlooked. This is especially likely to happen when the localized symptoms, such as pain and tympanites, are very slight. Sometimes the only things which call attention to the onset of peritonitis are the rapid change in the general condition of the patient and in his pulse and temperature.

The diagnosis of localized peritonitis also is often difficult. Many obscure, severe, febrile conditions are shown by the autopsy to be deep-seated suppurations in the abdominal cavity. We should never neglect taking a careful history and making a thorough examination, including rectal and vaginal palpation and exploratory puncture.

It is well to remember that even a pregnant uterus and a distended and therefore painful bladder have been mistaken for peritonitis! Hysterical meteorism (see the chapter on Hysteria) may also be erroneously ascribed to peritonitis.

Treatment.—Although severe cases are generally almost hopeless, yet we must try to meet the symptomatic indications, and must do all in our power to promote a limitation of the process, if it be possible.

External counterirritants or "revulsants" are seldom of much use. Painting with tincture of iodin and mercurial inunctions seem so utterly purposeless that they should be discarded. The local abstraction of blood cannot be employed in an extensive peritonitis with constitutional prostration. It is

only in a circumscribed peritonitis which is very painful, and when the general condition of the patient remains comparatively favorable, that bleeding is to be considered. Under these circumstances the application of eight to fifteen leeches sometimes causes decided abatement of the pain. The local application of ice to the abdomen is universally in vogue. It moderates the pain, and it may also have a beneficial influence in quieting peristalsis. Still, some patients cannot bear ice, and sometimes hot cloths and poultices give great relief.

Of all internal remedies, there is but one of great value, namely, opium. This in large doses (15 to 20 drops of laudanum or 1 gr. [gm. 0.05] of extract of opium every one or two hours) almost always proves beneficial. It moderates both the pain and the vomiting or eructations; and also, by diminishing the peristaltic movements of the intestine, opium contributes in another way to assuage the suffering and possibly to limit the spread of the inflammation. Experience shows that almost all patients bear large doses of opium remarkably well in peritonitis. Perhaps this is because the drug is only slowly absorbed. To substitute injections of morphin for opium is wise only in cases where we wish to produce narcosis as rapidly as possible, or when the vomiting or pain does not prove amenable to the ordinary treatment. In such cases we may also prescribe laudanum by enema or the extract in a suppository.

Sometimes particular symptoms demand special attention. For vomiting we may employ, besides opium, bits of ice, small quantities of sherbet, and perhaps chloroform or cocain. If tympanites is excessive, we may try to remove some of the gas through a rectal tube passed as high up as possible. Turpentine enemata, containing 1 teaspoonful of turpentine to 7 ounces (200 c.c.) of a thin gum-arabic solution, are also recommended. Some physicians also puncture the distended intestinal coils with a fine trocar. Collapse and cardiac failure require the exhibition of stimulants, such as camphor oil subcutaneously, ether, champagne, etc. A hypodermoclysis of normal (0.6 per cent) saline solution is also of service. It is generally very difficult to nourish the patient. As a rule, small quantities of ice-cold milk are the best of anything as food.

The surgical treatment of acute peritonitis is constantly growing in importance. It is self-evident that in the case of a circumscribed abscess in the abdominal cavity the only way of giving prompt relief is by operative interference; but even in acute diffuse peritonitis the results obtained by laparotomy, while not brilliant, are encouraging, and further trials are the more imperative, because otherwise the prognosis of such cases is almost absolutely hopeless. The more promptly the inflammatory germs and septic matter are removed from the abdominal cavity, the greater is the possibility of permanent cure. The details of the surgical treatment of peritonitis must be sought elsewhere.

CHAPTER II

CHRONIC AND TUBERCULOUS PERITONITIS

Ætiology.—Chronic nontuberculous peritonitis is a rather rare disease. It is found most frequently in post-mortem examinations of patients who have

had for a long time ascites due to venous stasis, and on whom repeated punctures have been practiced. The chronic peritonitis, however, is not the direct result of the passive hyperæmia in such cases, but is, as already hinted, due in most instances to the puncturing of the abdomen during life for the removal of the ascitic fluid. Exceptionally a chronic peritonitis occurs as a sequel to some severe intestinal disorder, such as ulceration. Thus, chronic peritonitis is sometimes observed to follow typhoid fever.

Chronic peritonitis may furthermore be the result of an acute peritonitis. The latter seldom terminates in this way, but still it may, when rather mild and not quickly fatal. The encapsulated exudations of peritonitis usually persist a long while, as was implied in the preceding chapter.

There is still great uncertainty about the ætiology of those cases which start in a subacute or chronic manner, without special cause. Sometimes they seem to be due to an injury of the abdomen; in other cases there are special constitutional influences, such as alcoholism, which may occasion the disease. In children, and less often in adults, we sometimes see cases of "simple exudative peritonitis," with a serous exudation. It is possible that in such instances pathogenic organisms of various kinds occasion the inflammation; but in order of frequency there is scarcely a doubt that tuberculosis should be named first. Just as in pleurisy, many cases of what is apparently primary "simple" peritonitis turn out eventually to be tuberculous.

The tuberculous is the most frequent form of chronic peritonitis. It is often merely a part of the tuberculosis of serous membranes in general (*vide* pages 352 and 448), of which mention has been already repeatedly made. In these cases it is usually due to a conveyance of the process from the pleura through the diaphragm. Another way in which tuberculous peritonitis may arise is by infection from neighboring tubercular organs. Tubercular intestinal ulcers are among the chief causes of this kind, the ulcer extending to the peritoneum; or the peritonitis may be excited by tuberculous retroperitoneal or mesenteric lymph-glands. In women tuberculous peritonitis may be developed in consequence of tuberculosis of the genital organs. Tuberculosis of the uterus sometimes affects the Fallopian tubes by direct extension, and thence the virus enters the abdominal cavity and excites its specific inflammation. In conclusion, we have to mention that, in general miliary tuberculosis, the peritoneum also may be the seat of numerous tubercles, although these do not, as a rule, give rise to important symptoms.

Pathology.—In severe cases of chronic peritonitis—and scarcely any others have come to autopsy—the peritoneum is usually found to be considerably thickened. The intestinal coils are joined to one another and to the neighboring organs by numerous and extensive adhesions. The false membranes are often delicate and easily separable, but in some cases it is a hard matter to disentangle the confused mass into which the intestines have been rolled. Sometimes the liver and spleen are covered by firm, tough capsules. The omentum and mesentery are much shrunken, hence the name *peritonitis deformans*. The omentum may indeed be transformed into a single thick cord. As a rule, there is little liquid effusion, and perhaps none. In simple chronic peritonitis, such fluid as may be present is usually a cloudy serum, sero-pus being seldom seen.

There are sometimes many clots of fibrin suspended in the fluid. If the

abdomen has been repeatedly punctured during the life of the patient, we often may recognize the separate punctures, on the inner surface of the peritoneum, from the hemorrhages, adhesions, or similar changes which have occurred. In rare instances a peculiar form of chronic peritonitis has been observed as a sequel to punctures for ascites, called by Friedreich "chronic hemorrhagic peritonitis with hematoma." In it almost the entire peritoneum is covered by a newly formed membrane permeated with large ecchymoses.

We will also mention briefly a peculiar form of chronic peritonitis, which has not yet been sufficiently investigated. This leads to the formation of numerous small nodules in the peritoneum, which are usually at first regarded as tubercles, but upon microscopic examination are found to be nodules of connective tissue.

Tuberculous disease of the peritoneum may be divided into two forms: tuberculosis of the peritoneum, which may be acute or chronic, and tuberculous peritonitis, which is usually chronic. In tuberculosis the peritoneum is covered with numerous tubercular nodules, varying in size from a millet seed up to a pea; but there is not much coincident inflammatory change. In genuine tuberculous peritonitis, on the other hand, the inflammatory changes above described are well marked, while sometimes it requires a microscopic examination to demonstrate the tuberculous nature of the inflammation, by the detection of tubercles and cheesy degeneration in the newly formed tissue. Tuberculous peritonitis is usually rather chronic, so that the adhesions are numerous and strong. The amount of liquid effusion varies, being sometimes considerable and sometimes scanty. Just as in tuberculous pleurisy, it is not rare for the exudation to be bloody. In long-continued peritoneal tuberculosis the omentum is found irregularly thickened, and the whole intestinal cavity filled with tumor-like masses (lymph-glands, etc.). Occasionally localized tuberculous disease of the peritoneum occurs. The ileocæcal tuberculosis, previously referred to in the chapter on intestinal tuberculosis, belongs primarily in this category. Among the more important associated conditions we have tuberculosis of other organs, and also the comparatively frequent combination of tuberculous peritonitis with hepatic cirrhosis (*q. v.*).

Clinical History. Diagnosis.—If an acute peritonitis becomes chronic, the violent symptoms gradually abate, while another group of symptoms takes their place. In other cases the chronic disease develops gradually and insidiously.

The sensitiveness of the abdomen is never so extreme as in the acute inflammation. Sometimes, to be sure, the patient complains of dull pains and a sense of abdominal oppression, but quite often the pain is either constantly or at times insignificant. On physical examination we usually find moderate distention of the abdomen. Frequently this is not perfectly uniform, certain coils of intestine being especially prominent. Occasionally there is no abdominal distention whatever, the belly is flat or concave, and the walls are tense and unyielding. The abdomen is more distended if there is a large amount of liquid exudation or if there is extensive tuberculous new growth.

In many instances palpation furnishes very characteristic signs, for sometimes the thickening of the omentum and the numerous fibrous interintestinal bands above described can be felt through the abdominal walls as peculiarly resistant masses or uneven prominences. Indeed, if the omentum is rolled

up it may closely simulate a new growth. The same is true of enlarged lymph-glands. Not infrequently, particularly in tuberculous peritonitis, the liver is enlarged so that its lower edge can be felt. But in other cases of chronic peritonitis there are no changes discoverable by palpation, or they may be concealed by an effusion or by the tenseness of the abdominal walls. A large exudation can be demonstrated by the great distention, or by its causing fluctuation, or by the signs yielded on percussion. As a result of the adhesions of the intestines to one another, we find that the fluid does not change its position very readily upon change of position of the patient. This circumstance is indeed one means of differentiating between peritonitic exudation and ordinary ascites. Not infrequently the resonance upon percussion is very greatly impaired, even when there is no large amount of fluid exudation. This is probably due to thickening of the peritoneum, fibrinous deposits, and similar changes. Peritonitic friction sounds are sometimes audible, particularly in the hepatic region. It has been already stated that the distortions and flexions which the intestines may undergo in chronic peritonitis may result in obstruction. In the same way the duodenum or the ductus choledochus may be so occluded as to occasion persistent jaundice.

The objective signs of both the simple and tuberculous forms of chronic peritonitis have been embraced in one description, because the abdominal signs of the two are identical. To differentiate between them, other factors must be considered. We regard the patient's constitution and general appearance, and inquire into his family history, or discover if there are other ætiological factors, such as previous tuberculous disease. A careful thoracic examination is extremely important. If we find the signs of coincident pulmonary tuberculosis, or of pleurisy, then it is almost indubitable that the peritonitis is tuberculous. The course of the fever and pulse is very important. Persistent hectic fever and persistent increasing frequency of the pulse with moderate fever must always arouse strong suspicions of tuberculosis. The character of the exudation obtained by aspiration may be significant, for the admixture of blood and a lymphocytosis occur mainly in tuberculous peritonitis, but we may also have serous exudation in tuberculous peritonitis, just as in the ordinary tuberculous pleurisy. Tubercle bacilli are not usually present in the exudation of tuberculous peritonitis.

To diagnosticate simple tuberculosis of the peritoneum, when not attended by marked inflammatory changes, is generally a difficult matter. Often it is absolutely impossible. Frequently there is no abdominal pain or tenderness whatever. The abdomen is usually but moderately distended, as a result of the effusion present. If we find such a condition in a patient who is known to have some other tuberculous affection, we are justified in suspecting that there is a simple tuberculosis of the peritoneum.

It is not unusual to observe the combination of hepatic cirrhosis with peritonitis, which we have already briefly mentioned. We have then splenic tumor and the other signs of portal congestion, in addition to the symptoms of chronic peritonitis, such as pain and fever. The amount of fluid in the abdomen is usually considerable. As a rule, the patients are hard drinkers. The hepatic cirrhosis may be the primary disease, and if so, it joins with the constitutional results of alcoholic excess in promoting the liability to tuber-

culous infection. We have also observed a combination of a syphilitic atrophic liver and tuberculous peritonitis.

Particular notice should be given to the chronic peritonitis of children, already mentioned. The occurrence of ascites in children between the ages of two and ten years has been observed repeatedly, both by other authors and by ourselves. The ascites, which may be considerable, cannot be traced to any cause, and after a few months completely disappears. The child during this time is usually rather pale and languid, but not much emaciated, nor does he suffer great local discomfort. There may be no fever. Since the cases often recover, their pathological anatomy remains obscure. Probably they are a mild form of simple chronic peritonitis. Still, of course, there may be other causes for ascites, such as hereditary syphilitic disease of the liver.

In children tuberculous peritonitis plays an important part in general tuberculosis of the abdominal organs, a condition formerly known as *tabes mesenterica*. In these cases the tuberculosis probably originates, as we have already said, in the intestine, so that usually we find the intestine, peritoneum, liver, and abdominal lymph-glands all simultaneously involved. The clinical symptoms are often due mainly to the peritonitis. The abdomen is distended and painful, and there is an effusion. Often there is also obstinate diarrhea, as a result of tubercular intestinal ulcers, with persistent fever of an intermittent character, emaciation, and anæmia. The tubercular process may eventually involve the lungs, pleura, meninges, and other organs, or it may never extend beyond the abdomen.

As to the course of chronic peritonitis we have little to say. The simple chronic peritonitis may terminate in recovery, although on account of other coexisting lesions this event is rare, except in the special form which children present. Many cases of tuberculous peritonitis prove fatal in a few months or weeks. The fatal termination of peritoneal tuberculosis may be occasioned by a septic peritonitis, due to perforation. We have seen two cases of this sort in which the tuberculous focus perforated the intestinal wall from its outer side. In many instances, however, chronic tuberculous peritonitis has a favorable issue, or at least there is very great abatement of all symptoms. This is particularly apt to be the case in what is called primary tuberculosis of the serous membranes in general (*vide supra*). If, in this disease, there is no simultaneous tuberculosis of the lungs, intestines, or other organs, then the final reabsorption of the exudation is possible, just as in tuberculous pleurisy. It must be confessed that often the recovery is not permanent, for the tubercles may appear later in some other part of the body.

Treatment.—The means by which we can exercise a favorable influence upon the course of chronic peritonitis are scanty. Nevertheless, very satisfactory results can be obtained in some cases by careful treatment. Great weight should be laid, above all, upon the constitutional treatment (continuous fresh air and rest cures, if necessary, in suitable health resorts or sanatoria, careful nourishment, etc.). Besides this, certain local remedies act favorably, especially the methodical inunction of the abdomen with green soap (spiritus saponatokalinus, or a salve containing equal parts of green soap and simple ointment). Alcohol compresses (thirty per cent), hot applications, Priessnitz applications, are also often of use. Inunctions of blue ointment, iothion salve, etc., may also be tried. Internal remedies are usually only indicated as symp-

tomatic therapy (opium, cathartics, etc.). If there is considerable exudation, diuretin, acetate of potassium, and similar diuretic remedies are indicated. Whether curative results are to be expected from tuberculin further experience must determine.

Of late the surgical treatment of peritoneal tuberculosis has had very favorable results. It has been observed that in many cases of tuberculous peritonitis, laparotomy, with as complete an evacuation of the exudation as possible (better than can be accomplished by mere puncture of the abdomen), exercises a wonderfully beneficial influence upon the course of the disease, and even seems in many cases to occasion complete recovery. This fact is interesting, although not yet wholly explained. Further observations must be collected with regard to this procedure, but our own experience justifies us in recommending it in suitable cases. Particulars with regard to its employment are taught in works on surgery.

CHAPTER III

ASCITES

(Hydroperitoneum)

The name ascites is given to a collection of transuded serum in the abdominal cavity due to venous stasis. The peritoneal veins belong to the portal system, so that among the diseases which lead to ascites those which impede the portal circulation are chief. As we shall see in the next section, ascites is, therefore, of frequent occurrence in cirrhosis of the liver, syphilitic disease of the liver, compression of the portal vein by tumors, thrombosis of the portal vein, and similar disorders. Ascites is also frequently present as one of the dropsical symptoms in the general circulatory disturbances, of cardiac disease, and in the course of various acute and chronic renal affections.

The clinical significance of ascites is due partly to the local discomfort occasioned by the presence of any considerable amount of fluid within the abdominal cavity. Small quantities of serum are often unnoticed by the patient; but, where many quarts (15 to 20, or even more) of transudation exist, the abdominal walls become greatly distended, and the patient has a very troublesome feeling of pressure, weight, and tension. What is of still greater importance is the crowding upward of the diaphragm. Respiration is thereby not a little impeded. If the ascites is great, the lower lobes of the lungs are so compressed that a considerable degree of atelectasis is produced.

To demonstrate ascites by physical examination is possible only when a considerable accumulation exists. Then the belly is prominent, its walls are tense and shining, and, the base of the thorax being gradually distended by the pressure of the liquid, the lower part of the thorax seems much broader than the upper. Distended veins are usually visible through the skin of the abdomen, like blue lines, here and there. These represent collaterals used for the obstructed venous circulation. As soon as the abdominal tension has attained a certain degree, fluctuation can be perceived by laying both hands upon the abdomen and imparting gentle but quick impulses to the fluid through the

walls. Percussion gives a dull sound everywhere that the fluid is in contact with the abdominal walls. Gravity, of course, leads the liquid to occupy the dependent parts. In the dorsal decubitus, and when the transudation is of medium amount, the dullness is bounded in the central and upper parts of the abdomen from a region of tympanitic resonance by a line concave toward the head of the patient. The surface of the liquid being horizontal, of course the dullness extends higher along the sides of the abdomen, while the tympanitic resonance is present in the central part. We would add that, where the layer of ascitic fluid is thin, we can obtain dullness only by light, superficial percussion. If the plexímeter or finger is pressed deeply in, the fluid is crowded to one side, and we get a tympanitic sound from the underlying coils of intestine. A factor of great diagnostic value is the change of dullness on change of position of the patient. If he lies upon one side, the fluid seeks the dependent portions of the cavity, and gives rise to extensive dullness there, while the opposite side now yields a tympanitic resonance. Or, if he changes to the other side, it in turn becomes dull, and the side previously dull becomes tympanitic. Similar differences are found between the results of percussion in a horizontal and in a sitting posture. It is only when the accumulation is very abundant that there is dullness over the entire abdomen.

The signs mentioned enable us in most cases to make a diagnosis of ascites with ease and certainty. It is, indeed, not always easy to distinguish a transudation of serum from the exudation of chronic peritonitis, for, of course, either sort of fluid would yield the same physical signs. Only, the change in the area of dullness consequent upon a change of position is less pronounced in case of an exudation, because the peritonitic adhesions impede the movements of the fluid; and we have, besides, all the other symptoms to guide us: there may be pain, fever, or thickening of the peritoneum discoverable on palpation, or signs of tuberculosis; or, on the other hand, there may be some cardiac or hepatic disease which would render ascites probable. If the fluid is drawn off, its character will sometimes aid us in diagnosis. Ascites yields pure serum, containing almost no morphological constituents. Its specific gravity is usually less than that of a peritonitic exudation, because it contains less albumen. We may say that the specific gravity of the fluid found in peritonitis is generally above 1.018, and that of ascites about 1.012, or even lower. The albumen content of ascitic fluid is about one to three per cent, while that of peritonitic exudate is as high as four to six per cent. Hemorrhagic ascites sometimes occurs in anæmic patients who are suffering from marked portal obstruction, as we have ourselves seen, for example, in hepatic syphilis.

Chylous ascites due to an abundance of fat in the fluid may appear as a result of occlusion of the thoracic duct, but it should be pointed out that ascitic fluid has sometimes a chylous opacity, even when it contains no fat (emulsion of albuminous substances?).

There may be equal difficulty in the exclusion of ovarian cysts, particularly since the cysts are sometimes so large as to fill the whole abdominal cavity. We must first map out accurately the dullness on percussion, and also see if it varies with changes of position. In cases of ovarian tumor, change of position does not make much difference. The resonance on percussion of the deepest and most dependent portions of the abdomen may be misleading, in this way, that even in ascites a narrow zone here may be tympanitic. This should be

remembered. Thus, just above the symphysis, there is sometimes a tympanitic resonance in ascites which might readily be mistaken for a proof of the existence of an ovarian tumor. The explanation is that in the places indicated a coil of intestine with a short mesenteric attachment may remain in contact with the abdominal wall in spite of ascitic accumulations. Further aid in the differential diagnosis is to be obtained from the history of the case (place where the swelling began), from a consideration of possible causative diseases, and from a vaginal examination, including direct palpation of the ovaries. In ascites the uterus is freely movable, while in case of ovarian tumors it is often bound down by adhesions. Further particulars may be sought in books on gynecology.

Treatment.—The treatment of ascites depends largely upon the disease of which it is a symptom. As to the symptomatic treatment of ascites itself, we will confine ourselves to a few words about tapping. This operation is indicated when the local disturbances caused by the ascites are great; that is, if there is an unbearable sensation of pressure and tension, and, above all, if the crowding up of the diaphragm causes much dyspnœa. The instrument to be used is a common trocar of medium size. If strength permits, the puncture can be most conveniently done with the patient sitting in a chair, or crosswise on the edge of the bed. We usually choose as the best place for the puncture a point in the left lower part of the abdomen, or, if the patient is seated, one in the linea alba, about halfway between the navel and the symphysis. Of course we should first see that the bladder has been emptied. In the majority of cases we are obliged to make the puncture with the patient in the lateral position; we then insert the instrument in the lower and left side of the abdomen, about halfway between the navel and the anterior superior spine. The exudation should never be allowed to escape too rapidly. We may permit large amounts of liquid (5 or 10 quarts, or more) to flow slowly away at one tapping. We close the puncture with sterile cotton and adhesive plaster, or with iodoform collodion. Often the fluid trickles out through the opening, because the abdominal walls have lost their elasticity on account of the persistent distention. We may then employ a suture to close it. After tapping, the laxness of the walls is favorable to palpation of the abdominal organs.

Inasmuch as tapping does not remove the cause, there is in most cases a very rapid reaccumulation of fluid. Thus the system is deprived of much albumen, and nutrition is impaired, so that not infrequently the operation is followed by decided loss of strength. Therefore, we should not tap in ascites, as a rule, unless the indications for the operation are urgent.

[If, as is very frequently the case, the fluid continues to drain away through the puncture after the trocar is withdrawn, good rather than harm results, provided the danger of irritation of the skin and of bedsores is kept in mind and guarded against, and an instrument of moderate size is used.

Flint advocates early and repeated tappings if the fluid causes discomfort and does not yield to diuretics or cathartics. The pressure is removed in a measure from the abdominal and thoracic organs, and nutrition is thus promoted. The fluid is likely to return, but it does not always do so, or it may do so only slowly. He reports cases in which, after repeated removal, the fluid ceased to return and the patient remained apparently well. The result must

depend, of course, chiefly on the underlying cause, which is sometimes very obscure.

In cases of cirrhosis the same principles govern Flint's treatment.]

CHAPTER IV

CANCER OF THE PERITONEUM

Carcinoma is the only new growth of any practical importance to which the peritoneum is liable. Primary endothelial cancer, analogous to the growth which attacks the pleura, is very rare. Cancerous growths here are usually secondary to cancer of the stomach, intestine, pancreas, liver, or some other organ. Often the secondary nodules are numerous, and almost as small as peas, presenting what is called miliary carcinosis of the peritoneum. They cover the omentum, mesentery, and parietal peritoneum. Separate nodules of larger size are less frequent. These may be found in the omentum, in Douglas's pouch, around the navel, or in other situations. Colloid cancer attains the most diffuse and extensive development of any variety. The retroperitoneal lymph-glands may also present at the same time large cancerous growths. Often the development of cancer in the peritoneum is attended with pronounced inflammatory disturbances—that is, we have a cancerous peritonitis.

Symptoms.—The symptoms of peritoneal cancer resemble in many points those of chronic tuberculous peritonitis. Simple miliary carcinosis may be very insidious and give rise to no special symptoms, so that it often is unsuspected. In many cases a moderate amount of fluid collects in the abdomen, and this, if we are aware of the existence of a primary cancerous growth, may lead us to surmise a secondary peritoneal carcinosis. The symptoms are much more pronounced if there is cancerous peritonitis. In that case there is usually very severe pain, marked abdominal distention, and constipation. We may sometimes feel the larger nodules in the omentum or upon the inner surface of the anterior wall of the abdomen, or even those in the lowest part of the abdomen, by palpation through the vagina or the rectum. We have repeatedly observed the development of nodules in the navel in carcinosis of the peritoneum, which could be distinctly felt externally as an infiltrated edge of the umbilicus. We have repeatedly felt a hard round strand running down the linea alba from the navel—probably a carcinomatous infiltrated lymph-vessel. The exuded fluid in the abdominal cavity is sometimes merely serous, but it may be hemorrhagic. When the new growth has been diffuse, and particularly in case of colloid cancer, the exudation has repeatedly been found to present a milky opacity. Sometimes this fluid also has been tinged with blood. The opacity is due to fat, from fatty-degenerated and disintegrated cancer cells. Occasionally the microscope reveals characteristic cancerous elements in the fluid. [Diagnosis based upon the microscope alone has repeatedly proved to be erroneous.—V.]

Diagnosis.—The diagnosis cannot be made with any positiveness unless, as a sequel to a primary cancerous growth already demonstrated, we observe the evident tokens of peritoneal disturbance, such as free fluid, pain, points of resistance on palpation, and distention of the abdomen. Other factors are

the patient's age, cancerous cachexia, and secondary glandular enlargements, particularly in the groins and supraclavicular spaces.

Treatment.—Treatment must be confined to efforts at mitigation of the suffering. Warm applications and morphin are chiefly employed. A puncture may be indicated in marked distention of the abdominal walls.

SECTION VII

Diseases of the Liver, Bile Ducts, and Portal Vein

CHAPTER I

CATARRHAL JAUNDICE

(Icterus catarrhalis. Gastroduodenal Catarrh with Jaundice)

Ætiology and Pathological Anatomy.—Catarrhal jaundice is still the almost universal term applied to the most common form of jaundice. It develops in previously healthy individuals, usually in a rather acute manner, and sooner or later (as a rule, after some weeks) terminates in complete recovery. There can be scarcely any doubt that a simple catarrhal inflammatory process in the bile ducts is the cause of this not infrequent clinical picture. We do not know exactly by which infectious agent the catarrh is produced, nor where it begins and how far it extends, inasmuch as the benignancy of the disease hardly ever affords an opportunity for anatomical investigations. It is not improbable that the infectious material enters the larger bile ducts from the duodenum. It has not been proved, however (at least not for all cases), that the development of the jaundice is always preceded by an actual catarrh of the stomach and the duodenal mucous membrane. This was formerly assumed, and the term "gastroduodenal jaundice" applied. This can be decided only by a study of the clinical course. It is certain at least that a more or less complete occlusion of the common bile duct or even the other bile ducts does occur. This is due to a swelling of the mucous membrane, and perhaps also to the mucus itself and the desquamated epithelial cells. The occlusion of the bile ducts leads to biliary obstruction in the liver, and this to jaundice (*vide infra*).

Opposed to this explanation of "catarrhal jaundice," which in my opinion is the most natural and probable, a number of authors have lately proposed another theory. According to this, the jaundice should not be regarded as an "obstructive jaundice," but as a result of a disturbed function of the liver cells ("paracholia"), so that the bile is no longer secreted into the smallest bile ducts, but directly into the blood and lymph capillaries. In our opinion, this assumption is worthy perhaps of further investigation in certain other forms of jaundice that occur after acute intoxications. For ordinary catarrhal jaundice, however, it appears to us much too artificial, and by no means satisfactory. We believe that the assumption of an infectious toxic disease of the mucous membrane is much more plausible. Naturally, it is not certain whether

the same infectious agent, such as the colon bacillus, is always the exciting cause, or whether there are a variety of organisms. The rather frequent epidemic and endemic occurrence of catarrhal jaundice that has been observed is most instructive. This undoubtedly points to a definite infectious cause. Rather extensive epidemics of jaundice have been repeatedly observed in barracks, prisons, and communities in a manner to be explained only by the supposition of some local source of infection. We once observed a small, benign epidemic of jaundice among many members of a student fraternity. In this instance the beer that had been drunk was held responsible. In some instances the epidemics of jaundice have followed revaccination, obliging one to think of the transfer of the pathogenic organisms by means of inoculation. As a rule, to be sure, jaundice occurs sporadically, and it is not usually possible to demonstrate any special cause for the disease, except that there is often a connection with some marked error in diet, with catching cold, or some mental excitement, such as anger. Catarrhal jaundice is observed chiefly in youthful individuals; sex has no marked influence.

Symptoms and Clinical Course.—Very frequently, though not invariably, there are, for some days before the appearance of the jaundice, definite signs of gastrointestinal disturbance, as well as certain constitutional symptoms. Obstinate vomiting, marked pain in the stomach, and similar symptoms are not infrequent. Very often the trouble begins with loss of appetite, a bad taste in the mouth, nausea, a sense of pressure in the epigastrium, eructations, and constipation, accompanied by languor. Soon after, the patient himself notices the yellow color of the skin, or his attention is called to it by those about him.

We still hold provisionally to the view that the cause of catarrhal jaundice is mechanical; and we desire, with a view to preventing repetition, to describe here with some minuteness those symptoms which occur in varying severity in all cases of obstructive jaundice.

The bile is secreted in the liver under an extremely low pressure, and consequently a comparatively slight obstruction in the bile ducts suffices to prevent the discharge of bile into the intestine. In ordinary catarrhal jaundice, and also in other varieties of jaundice, the obstruction to the flow of bile is seldom complete, and if so, only for a time. Still, a considerable amount of bile collects, and distends even the intrahepatic ducts. As soon as this stasis has reached a certain point, the stagnant bile is absorbed mainly by the hepatic lymph-vessels, and perhaps also directly by the blood vessels of the liver. Thus the bile and all its constituents are poured into the blood and carried to all parts of the body. No more than a few days need elapse before the bile pigments are absorbed into the tissues, and give rise to the evident yellow color of the skin and visible mucous membranes which we call jaundice. Usually the yellowness of the conjunctiva is the first thing to attract attention. Later the entire skin becomes yellow, and the same color is plainly visible in the mucous membrane of the mouth and throat, especially after we have produced temporary anæmia by pressure, as in the lips. The intensity of the yellow discoloration presents the most varying degrees. With artificial illumination (with the exception of the white electric light) the icteric coloring of the skin is scarcely noticeable. The internal organs, which we cannot see, are likewise stained to a greater or lesser extent. Any abnormal collection of liquid will

also have a marked yellow color. The cornea, the peripheral nerves, and the cartilages alone escape unstained. In other parts we may not only find this diffuse impregnation with the biliary pigments, but even solid granules of the latter.

A jaundiced patient often presents other indications of the presence of biliary coloring matter than the color of his skin. There is often an itching of the skin, which may be very troublesome. It may be so bad at night as to disturb sleep. The scratching thus induced often causes numerous excoriations and fissures, which may even occasion quite large furuncles. Urticaria is also sometimes observed. The cause of the marked irritation of the sensory nerves of the skin is not quite clear. Sometimes, in spite of the most extreme jaundice, the itching of the skin is entirely absent; in other cases, even with slight icterus, it is very marked and distressing. A peculiar disease of the skin, which has been described in connection with chronic jaundice, is called *xanthelasma* or xanthoma. It presents bright-yellow spots, usually somewhat elevated, which look like yellow fat droplets and are found mainly on the eyelids, though present also on other parts of the body.

The remaining symptoms of hepatogenous jaundice may be divided into two groups. The first group comprises the symptoms excited by the presence of the biliary constituents, and particularly of the biliary acids, in the blood, while the second group is due to the lack of bile in the intestinal canal.

We have seen that, when the biliary outlets are occluded or narrowed, the constituents of the bile are absorbed. We have already learned in part what becomes of the bile pigment thus conveyed into the blood vessels. That it also displays poisonous properties has not yet been absolutely proved. The presence of the bile acids in the blood at any rate is of considerable clinical importance. Physiology has shown that these acids possess certain injurious qualities, and, among others, the power to destroy red blood corpuscles. But in reality few if any blood corpuscles are destroyed by the bile acids in the blood, because they are too much diluted, and besides, seem in large part to be quickly decomposed after absorption. These acids do really, however, excite certain nervous centers in a way to give rise to decided clinical symptoms. The most frequent effect is that produced by the cholate of sodium upon the cardiac ganglia, or possibly also upon the center for the vagus, and it is evinced by a slowing of the pulse. This is an almost invariable phenomenon, provided there be no fever or other complication, and is seen not only in simple catarrhal jaundice but in all cases of hepatogenous icterus. The pulse rate is from 64 to 50, or even less. Slight irregularity in the heart's action is not infrequent. There are certain other nervous disturbances often seen in jaundice, referable to the presence in the blood of biliary constituents, and in particular of biliary acids. Sometimes there is a striking languor and muscular weakness, or headache, insomnia, or the patient is "out of sorts." Grave nervous symptoms, sometimes seen in jaundice and grouped under the name of *cholæmia,* will be separately discussed later. It also deserves a brief mention here that many cases with marked jaundice have a noticeable tendency to bleeding—that is, a sort of "hemorrhagic diathesis" with hemorrhages into the skin and in the viscera, epistaxis and analogous occurrences.

We come now to a consideration of those symptoms which arise because the bile does not reach the intestine. In this regard the standpoint of modern physi-

ology is essentially different from the old views. Formerly the bile was valued as an important digestive secretion; now it is regarded by many merely as an excretion, containing certain end products of metabolism; but perhaps this view is extreme, for the very fact that the bile is discharged into the upper part of the intestine suggests that it has a certain significance in the processes to which the intestinal contents are subjected, and there is certainly one function of the bile which is indisputable—viz., that it promotes the absorption of fat. The bile both contributes to the emulsifying of fat, and also promotes the passage of the fat drops through the intestinal walls into the lacteals. Now, in hepatogenous icterus this work remains undone, as is shown by the fatty stools. From time immemorial the white clay-colored stools of jaundice have been well known, and are employed as the best measure of the completeness of biliary retention. The light color of the stools is due partly to the lack of biliary pigment, for it is that chiefly which imparts to normal feces their dark-brown color; but the characteristic white clay color is due exclusively to the presence of undigested fat in large amounts. If an icteric patient is put upon a diet, as free of fat as possible, the stools are by no means clay-colored, but light brown. Upon microscopic examination of the feces in jaundice, sheaf-like aggregations of crystals are almost invariably observed. These were formerly supposed to be tyrosin, but Oesterlein has shown them to be in reality lime or magnesia soap.

Besides this influence upon the absorption of fat, there have been two other properties ascribed to the bile, having apparent significance for the pathology of jaundice. In the first place, there is the antiseptic power of the bile; this was said to explain the fact that in patients with biliary obstruction the feces usually have a particularly bad odor, and also that there are often excessive tympanites and flatulence. Secondly, the bile was reputed to exert an influence upon intestinal peristalsis—hence, the constipation frequently seen in jaundice. Of late, however, authorities have had doubts about ascribing either of these properties to the bile. The foul odor of the feces may be associated with the imperfect digestion of fat, and the constipation with the change in the diet and with other circumstances.

We must mention one other point: If the obstruction to the discharge of bile is actually situated at the papilla duodenalis—and this was formerly regarded as certain in catarrhal jaundice—then the discharge of pancreatic juice must also be affected, and we must also consider the results of the exclusion of this secretion from the intestinal contents. The presence of fatty stools may very well be partly dependent upon this point. We will discuss later the various other consequences of a disordered pancreatic function.

We must now inquire what becomes of the absorbed bile. As to the biliary acids, we have already said that they probably undergo decomposition. Of the other constituents, including the taurin and cholesterin and the pigmentary matter, we know the fate of the last-named only—that is, we have learned how Nature seeks to rid herself of this foreign substance. As soon as the amount of bile pigment in the blood and tissues becomes considerable, excretory efforts are made, in which the kidneys take the chief share. Certain changes take place in the urine almost simultaneously with, or even frequently somewhat before, the first appearance of a jaundiced hue in the skin; and these changes are due to the urine containing excreted biliary coloring matter.

The urine of jaundice is generally recognizable from its color, which is dark brown, like beer. The foam caused by shaking it is not white, but decidedly yellow. A bit of white filter paper dipped in the urine is stained yellow. If the urine is shaken with chloroform in a test-tube, the chloroform dissolves the pigment, and, on being allowed to collect at the bottom of the tube, displays a decided yellow color. This is known as the "chloroform test." Another reaction which usually gives a satisfactory result, but not always, is Gmelin's. If urine containing bile pigment is slowly poured down the sides of a test-tube containing a few cubic centimeters of nitric acid, the zone between these two liquids exhibits a fine play of colors. The oxidizing effect of the acid upon the biliary pigment produces a number of colored rings, the highest and most characteristic of which is green; next comes blue, then violet and red. Gmelin's test often shows very prettily if one filters the urine and then adds a drop of nitric acid to what remains upon the moist filter paper. The characteristic colored rings form around this drop. Instead of nitric acid, we may use tincture of iodin, diluted with nine parts of water. If a small amount of this solution is poured upon urine containing bile, in a test-tube, there is usually developed a ring of a beautiful green color between the two fluids.

The biliary acids also may be detected in the urine of jaundice; but the process is somewhat tedious, and the knowledge gained is of no great practical importance.

The urine very often contains pathological elements which are the result of kidney involvement subsequent to the irritative effect of the excretion of the abnormal products in the system. Nothnagel was the first to describe minutely the icteric casts—that is, hyaline casts which usually have a yellow tinge and quite often are completely covered with dark-yellow granules or with epithelium. The urine may contain a little albumen also, but this is not constant. If we add acetic acid, there may be an opacity developed which is said to be due to nuclein.

[The presence or absence of albumen depends largely on the amount of the biliary constituents and on the length of time they continue in action on the kidneys; their effect on these organs is more or less that of an irritant.]

The sweat glands also take part in the excretion of bile pigment. The latter can be demonstrated in the perspiration of jaundiced persons, as well as in their urine. Not infrequently the patient's linen is colored yellow by the sweat. On the other hand, no bile pigment is found in the tears, saliva, gastric juice, milk, or the cerebro-spinal fluid.

Having now considered the phenomena common to all cases of hepatogenous icterus, we revert to the subject of simple catarrhal jaundice. The prodromal gastric symptoms usually last a few days, more rarely a week or two, when the skin becomes evidently jaundiced and the other results of the icterus are also seen. The urine grows dark with biliary pigment, the stools become light-colored and more or less clay-colored. The nervous system is not usually seriously deranged, but still most patients feel very languid, and have anorexia and a tendency to constipation. The pulse often becomes somewhat slower than normal, and sometimes the temperature also is subnormal, 97° or 98° F. (36° to 36.5° C.), but there may be slight fever.

In most cases the physical examination of the liver is of interest, the organ being enlarged from the retained bile. Accordingly, the lower boundary of

hepatic dullness usually extends the breadth of two or three fingers below the edge of the ribs, and not infrequently the lower margin of the organ can be plainly felt. Often the gall bladder is so distended, both by bile and possibly by the mucus which the bladder itself secretes, that it projects from under the edge of the liver. In such cases, as Gerhardt tells us, we may sometimes make out by percussion a convexity in the lower line of hepatic dullness, which corresponds to the gall bladder. If the abdominal walls are lax, we may feel the distended viscus. As a rule, there is not much distress in the hepatic region, although now and then there is a certain sensation of pressure or tension. I have occasionally found the spleen distinctly swollen in severe cases of catarrhal jaundice.

The symptoms depicted seldom last longer than a few weeks. Usually a patient who takes proper care of himself begins to feel better in even less time. The urine grows lighter-colored, the stools darker, and the pulse more rapid. The yellow color of the skin often remains visible for quite a while, although gradually diminishing, even after the patient feels perfectly well; but at last the jaundice disappears also and recovery is complete. Relapses are indeed possible, particularly after errors in diet, but they are rare.

The termination of catarrhal jaundice is, therefore, almost invariably favorable. The entire course of the disease occupies about three to six weeks, rarely a longer period. I have seen cases of icterus, to be sure, especially in children, which, on account of their benignancy and final satisfactory outcome, could only be classified as catarrhal icterus, but which lasted half a year and even longer. The general condition was little disturbed, but the liver was markedly swollen and the spleen was rather decidedly enlarged. It is a very exceptional occurrence, but one which we must always think of as possible, for this apparently mild and secure condition to be suddenly merged into the grave, pernicious variety of jaundice. (See the chapter on Acute Yellow Atrophy of the Liver and Pernicious Jaundice.)

Diagnosis.—Catarrhal jaundice is usually easily diagnosticated. The diagnosis is made chiefly from the course of the disease—the development of jaundice, preceded by gastrointestinal symptoms, in a previously healthy person, and generally in a youthful individual. It is very important to exclude other conditions which might occasion jaundice. We must consider, therefore, whether the history of the case suggests the presence of gallstones (pain, hepatic colic), and be vigilant in our physical examination to detect a possible cirrhosis or new growth. In the case of elderly patients, particularly, it is not rare for what was at first regarded as an attack of ordinary catarrhal jaundice eventually to disclose itself as a grave chronic disease, such as cancer of the gall bladder. We should not make a diagnosis of catarrhal jaundice until we have carefully weighed all the rational and objective signs.

Treatment.—Most cases of catarrhal jaundice terminate favorably and require no active treatment. Rest and prudence are indicated, and the diet should be carefully regulated. Fat must not be eaten, for, as we have seen, it is poorly assimilated, and only excites abnormal processes of decomposition in the intestinal canal. Lean meat, bread, soups, if not too rich, vegetables, cooked fruit, and lemonade or tamarind water are allowable. Milk [unless skimmed] is to be avoided, as a rule, because of the fat it contains, but yet it is often well borne. Alcohol is to be allowed very sparingly if at all.

We should also employ internal remedies to mitigate the assumed gastric catarrh. The various stomachic tonics are frequently prescribed. Rhubarb is a favorite drug. A very good medicine is Carlsbad water, or the artificial Carlsbad salts, of which latter the dose is half a tablespoonful to a tablespoonful, in a tumbler of warm water, before breakfast, or twice a day. The alkalies are said to have a favorable influence upon the gastrointestinal mucous membrane, and they are also laxative; that they furthermore promote the secretion of bile, as many assume, is doubtful; but there is no doubt that the flushing of the kidneys by an abundance of liquid is beneficial. If there is obstinate constipation we are obliged sometimes to resort to more powerful remedies, such as castor oil, calomel, or rhubarb.

Lately much enthusiasm has been displayed about the treatment of catarrhal jaundice by large enemata of cold water. The injections are said to overcome the biliary retention by exciting peristalsis, and possibly by also promoting the secretion of bile. Once a day a quart or two of water, at 60° to 70° F. (12° to 18° R.), is injected, and is retained as long as possible. The good effect is said to be observable in a few days, both in the general condition of the patient and in the diminished amount of bile pigment in the urine, as well as the darker color of the stools. Other physicians recommend injections of a pint to a quart (0.5 to 1 liter) of lukewarm water several times a day. We have tried both methods repeatedly without being able to convince ourselves that the course of the disease is thus rendered very different from that under simple dietetic management; still, one may try irrigations, especially if the case is obstinate. Whether there are any internal remedies which promote the secretion of bile is very doubtful. The drug which is most recommended for this purpose, and is much employed in catarrhal jaundice, is salicylate of sodium, in the dose of 8 to 15 gr. (gm. 0.5 to 1), several times a day. The biliary acids have also been tried, in the form of purified ox gall, and also salol, podophyllin, calomel, and many others. We believe that all these remedies may generally be dispensed with.

The effort has also been made to empty the gall bladder by manipulation. Gerhardt states that sometimes the distended viscus can not only be felt through the abdominal walls (*vide supra*), but it can be so firmly compressed as to squeeze its contents into the duodenum. Sometimes the obstruction is said to yield suddenly, as if a plug were driven out. This method has not been extensively adopted. It seems applicable only in a limited number of cases, and is probably not free from danger. The suggestion of external faradization as a means to stimulate the gall bladder to contract and discharge its contents appears to me to be entirely illusory.

Among symptoms, the troublesome itching deserves particular attention. The remedies which may be recommended for this are bathing with cold water, rubbing the skin with slices of lemon, or the application of a two-per-cent solution of carbolic acid, or of salicylic acid or thymol dissolved in alcohol (one to three per cent); also menthol (headache pencil) and choloroform mixed with olive oil. Sometimes temporary relief is afforded by a dose of antipyrin or salicylate of soda internally. We have sometimes found baths, to which were added 10 to 15 drops of a mixture containing menthol, ʒij (gm. 7.5), and spiritus mentholi crispati, ℥ij (50 c.c.), very useful.

APPENDIX

CERTAIN SPECIAL FORMS OF JAUNDICE

1. Acute Febrile Jaundices. Infectious Jaundice. Weil's Disease.—There is a peculiar acute infectious disease which was first described by Weil, and then more fully by Fiedler and others, which seems to be always associated with an acute infectious inflammation of the gall ducts, and, having jaundice for a symptom, it may properly be described in this connection.

The disease is most frequent in the months of summer. It attacks by preference young and middle-aged men. Fiedler was struck by the frequency with which butchers suffered from it. The symptoms usually begin suddenly. Extreme chilliness, fever, headache, and malaise are almost always present at the onset. Jaundice usually appears on the second day, or soon after, and it may become severe. Its immediate cause is doubtless obstruction of bile, for the stools are colorless and the urine contains an abundance of bile pigment. The constitutional symptoms remain for several days quite severe. The patient complains of violent headache, wakefulness, and vertigo. Sometimes there is evident stupor or mild delirium. Upon physical examination we not infrequently notice herpes on the lips, besides the icterus. The tongue is coated. There is nothing unusual about the lungs or heart, except that the pulse is apt to be quite rapid. The abdomen is not particularly distended. The liver is often enlarged, but not always. An acute splenic tumor is very often, but not invariably, present. There is usually diarrhea. Vomiting may occur. The urine almost invariably contains albumen, and, as a rule, we find blood, epithelium, and casts, indicating a considerable amount of nephritis. In a few cases there are anuria and uræmia. Finally, a very characteristic symptom is the violent pain in the muscles, particularly in the calves of the legs, of which most patients complain. Epistaxis and hemorrhages into the skin have also been repeatedly observed.

With these symptoms the disease persists for from five to eight days, during which period the fever is often very considerable. Temperatures of 105° to 107° F. (40° to 41° C.) are not rare. Then the fever falls by crisis or rapid lysis, although seldom with perfect regularity. At the same time the other symptoms abate also, and convalescence ensues after an illness of ten to fourteen days in all. Many of the milder cases recover promptly, while others are prolonged by various sequelæ and fresh exacerbations. An unfavorable termination seems to be exceptional: it may result from uræmia.

It is highly probable that this perfectly specific disease, the most important symptoms of which are, as we have seen, jaundice, fever, swelling of the spleen, albuminuria, and pain in the muscles, is an acute infectious process, with a special involvement of the biliary ducts; but we do not yet possess any precise information as to its ætiology. It seems, however, that bacteria belonging to the colon-bacillus group are most probably connected with its development, inasmuch as the serum of patients suffering from Weil's disease has been repeatedly found to have an agglutinating effect upon typhoid bacilli (Widal's reaction). Possibly we do not always have to deal with absolutely the same morbific process, but with several allied conditions. A sharp differentiation

of Weil's disease from simple catarrhal jaundice is likewise not possible, as the latter, as already mentioned, often presents individual features which point to an infectious origin (splenic tumor, slight elevations of temperature, etc.). We have also repeatedly observed cases of benign, but still quite severe, febrile jaundice, without albuminuria or muscle pains, but with continuous high temperatures. These could only be interpreted as cases of primary infectious cholangitis. One of these cases was complicated by a unilateral pyelitis, which also pointed to a colon-bacillus infection.

The *treatment* must be purely symptomatic. In the beginning calomel is useful. Later we prescribe Carlsbad salts, salicylate of sodium, stomachics, or the like. The headache is often relieved by an ice bag or antipyrin; the muscular pain by the inunction of chloroform oil. From the first, the diet must be such as not to aggravate the nephritis (milk, simple soups).

2. **Chronic Hereditary or Family Jaundice.**—The occurrence of a chronic jaundice in several members of the same family has been described by various authors (Murchison, Minkowski, etc.). The anatomical basis of this condition is still not clear. It is usually explained by a congenital anomaly of the bile ducts or liver cells. In some of these cases a decided splenic enlargement is found. Minkowski found a pronounced deposit of iron in the kidney, which points to an abnormal destruction of red blood cells.

3. **Icterus Gravidarum. Menstrual Jaundice.**—Certain isolated observations point to a peculiar, perhaps toxicogenic, relation between jaundice and the female sexual functions. The repeated occurrence of jaundice with each pregnancy has been observed by Brauer, and the occurrence of jaundice during menstruation—so-called menstrual jaundice—has also been described.

4. **Congenital Chronic Jaundice.**—This is a remarkable condition and only very rarely observed. It persists without much other disturbance throughout life, and depends probably upon a congenital anomaly of the bile ducts.

CHAPTER II

BILIARY CALCULI

(Hepatic Colic. Cholelithiasis)

Ætiology.—Gallstones are of very frequent occurrence; Riedel asserts that some two millions of the inhabitants of Germany have gallstones, although this estimate seems to be very high. Despite their frequency, we have little positive knowledge of their causation. We can merely state certain circumstances which in all probability favor the formation of these concretions.

Biliary retention certainly acts in this way, both directly and by leading to an increased consistence and increased concentration of the bile. As a result, certain constituents which were before held in solution are thrown down. And yet this cause, however potent (*vide supra*), cannot be regarded as the only one. The chemical composition of gallstones suggests that their formation must be preceded by certain abnormal chemical processes of decomposition and of transformation. We cannot otherwise explain why the constituents of gallstones should differ, as they do in many ways, from the mat-

ters which normal bile holds in solution. For example, the pigment in gallstones is never found unchanged, but invariably exists in composition with lime. Now, normal bile contains only a trace of lime, so that long ago Frerichs expressed the opinion that the lime comes from the mucous membrane of the gall bladder. It is an important fact that the cholesterin and probably also a portion of the pigmentary matters are held in solution in normal bile by the combination of sodium with the biliary acids which it contains. If this sodium salt were decomposed from any cause, the matters named would naturally be precipitated. The decomposition of the salts formed by the bile acids is greatly promoted if the bile acquires an acid reaction; but of the circumstances in which this last-mentioned change occurs we do not yet have any accurate knowledge. Lately Naunyn, after extensive investigations, has propounded a theory that the formation of calculi is due in most cases to a primary disease of the mucous membrane of the gall bladder and the bile ducts. The disease causes the destruction of great numbers of the epithelial cells of the mucous membrane, with a resultant production of cholesterin, and of a compound of lime with bilirubin. Thus are formed friable masses of detritus, which are gradually changed into gallstones, in the interior of which it is still possible at an early stage to demonstrate the original pultaceous mass. As the cholesterin and the compound of lime with bilirubin continue to form by crystallization, and fresh deposits of firm material takes place, the stone gradually grows harder and larger. Very often, also, there is added at this stage a deposit of carbonate of lime. The cause of the original lithogenous catarrh is not known. Perhaps the mere mechanical obstruction of bile is causative, perhaps infection. In particular, the *Bacterium coli commune*, which originates in the intestines, seems to be important—or at least, if not this, a very similar kind of bacillus. If the supposition were confirmed that infection of this sort is a factor, it would also be natural, as shown by what has been previously stated, to consider that such an infection would have an influence upon the bile itself.

We have rather more knowledge as to predisposing causes than about the chemical processes involved in the formation of gallstones. Age seems to be an important factor. The great majority of patients are over forty. Gallstones are much less frequent between twenty and forty years of age, although we have not very infrequently observed typical cholelithiasis in younger individuals, and even occasionally in children. One reason why elderly people are so liable to this trouble is said to be the senile weakness of the muscular fibers of the gall bladder. Thus stagnation and retention of bile are promoted.

Sex also has a decided influence. All authors agree that gallstones are more frequent in females than in males, the proportion being about three to two. An explanation of this fact has been sought in the sedentary life of women, and particularly in the mechanical effect of women's clothes and tight lacing impeding the outflow of bile. It is a fact which certainly deserves consideration that we are very apt to find gallstones and a corset liver in the same individual. Far more important, still, appears to us the influence of pregnancy. With comparative frequency the first attacks of colic appear during pregnancy or the puerperium. At any rate, gallstones are found in women who have borne children much oftener than in childless women.

Much has been said about certain peculiarities of constitution, in their

relation to the formation of gallstones; and it is asserted that the mode of life may favor their development—as when too much food is taken, particularly an excess of fat and meat, and when there is insufficient bodily exercise. In general, statements of this sort must be viewed with much suspicion; still, we cannot deny that cholelithiasis is seen with striking frequency in obese women, and that the trouble seems to be somewhat more common among the favored classes than among the poor. There often seems to be an hereditary predisposition to gallstones, which fact also points to constitutional causes.

It has been said that there is a relation between cholelithiasis and gout, arteriosclerosis, and other diseases. This is not at all certain, though worthy of note. It is an interesting fact that sometimes the same individual has gallstones and renal calculi.

In rare cases there may be disease of the liver and of the bile ducts themselves, leading to persistent obstruction, and thus causing gallstones.

The reversed relation is very much more important, namely, that gallstones may lead to secondary diseases of the liver, such as carcinoma, biliary cirrhosis, and suppurative hepatitis.

Occurrence and Chemical and Physical Properties of Gallstones.—The place where gallstones are most frequently formed and found is the gall bladder. We may find in it any number, from one or two up to a hundred and more. The size varies from that of a grain of sand to that of a hen's egg. The large stones may completely fill the gall bladder; and sometimes the smaller stones are numerous enough to fill it also. The stones usually lie free in the bladder, although exceptionally they may be found adherent to its walls. Rarely the bladder presents a diverticulum, in which a stone has been formed. The lining membrane of the viscus often presents quite a severe catarrhal inflammation. This may have preceded the formation of the stone (*vide supra*), but, on the other hand, it may have developed as a result of the mechanical irritation of the mucous membrane occasioned by the concretions or by secondary infectious influences. Very often the wall of the gall bladder and the neighboring tissues are affected by mild or even severe inflammatory processes, suppuration, adhesive peritonitis, necrosis, perforation, and similar lesions (*vide infra*).

Stones which are found in the larger bile ducts are not formed in them, but they have become wedged in them while on their way to the intestine. Sometimes, however, stones are formed in the liver itself ("hepatic calculi"). These are seldom primary, but are usually the result of biliary obstruction because of persistent stenosis of the common duct. Hepatic gallstones of this sort may reach a diameter of a half to one centimeter. In such cases the small intrahepatic bile ducts are usually a good deal widened; or occasionally they present niches in which the stones lie. As a rule, the hepatic parenchyma surrounding the stone is in a state of chronic or acute purulent inflammation (*vide infra*).

In form, gallstones vary infinitely. The smallest are irregular masses, well described by the name of "gall sand." The larger stones are more or less round, oval, or polyhedral. The polyhedra are usually due to the mutual rubbing and pressure of a number of stones upon one another. In color, the stones vary according to the amount of pigment they contain, from almost black or dark brown to a lighter greenish or bright-yellow shade. A fresh

gallstone always sinks in water; but when dry, gallstones contain air and generally float. On cross-section, the hard stones are found to be either homogeneous or composed of layers. As a rule, there is a nucleus, darkly pigmented, which is surrounded by a lighter-colored envelope, itself either made up of concentric layers or evidently crystalline. Often the outermost layers are still distinguishable as peculiar darker and harder strata. If we examine thin scales of gallstones microscopically we shall find in them (as in renal calculi) an organic cement which holds the minute crystals together (Posner).

As to chemical composition, gallstones are usually divided into several groups. By far the most frequent variety is made up of cholesterin and pigment (bilirubin) mixed together in greatly varying proportions. The pigment is in great part combined with lime. On the average, stones contain about seventy to eighty per cent of cholesterin. Besides these two chief ingredients, most stones also contain lime and magnesium. In color they are light or dark, according to the smaller or greater proportion of coloring matter they contain. Stones of pure cholesterin are less common. These are usually found singly, are soft, and often are almost transparent. Most cholesterin stones have a nucleus of pigment and lime in combination. Pure pigmentary concretions are rare, and are generally small, like coarse sand. A still greater rarity is a stone made up entirely of lime. Such a stone is small and very hard.

Anatomical and Clinical Results of Gallstones.—It is by no means rare for gallstones to be present even in considerable number and size without causing the slightest discomfort. This is proved by the fact that often at autopsy gallstones are found by chance, although there had never been a symptom suggesting their presence. In such cases it is evident that the concretions have formed very gradually in the gall bladder, and never have led to mechanical or inflammatory disturbance.

In other cases, probably very numerous, the gallstones occasion certain disturbances, but these are comparatively insignificant and not of such a character as to enable the physician to make an absolute diagnosis. Patients of this sort complain at times of slightly painful or disagreeable sensations in the stomach and liver, particularly after bodily exertion, and they may also have other symptoms, such as slight dyspepsia. The objective examination yields nothing positive. The physician suspects a gastric disorder, a floating kidney, or a neurosis. It is important that he should always in such cases, particularly in women, consider the possibility of gallstones; for it is possible, if there is an actual cholelithiasis, that at any time there may be severe symptoms, the correct interpretation of which will be rendered much easier if these mild prodromata have been duly appreciated.

In a third class of cases the gallstones cause severe, and sometimes actually fatal, illness. The symptoms may be very characteristic, so that they can be recognized easily and absolutely; but not infrequently the condition is difficult to interpret, and misleading.

The form of the disease which can be recognized most certainly and which has been longest known to clinicians is characterized by the occurrence of distinct and peculiar paroxysms. These are usually termed biliary colic. An attack of this sort may occur suddenly, while the patient is apparently in perfect health. More often, however, the first distinct attack is preceded for

a long time by mild disturbances (*vide supra*), although these latter often receive little attention, and are apt to be misinterpreted.

The typical attack of biliary colic begins either with absolute suddenness or after some slight prodromata, such as chilliness, nausea, and malaise. It may occur at any time, day or night. The comparatively frequent sudden appearance of the pains in the evening hours, or even during the night in sleep, is particularly characteristic. Usually there is no exciting cause to be discovered. Sometimes the paroxysm is preceded by a slight injury or some other harmful influence, such as errors in diet, mental excitement, and the like. The chief symptom of the attack is the pain. This may be extreme from the start, or it may begin mildly and then rapidly or slowly become extreme. Usually the patient refers the pain chiefly to the epigastrium and to the right hypochondrium, but from here it radiates into the left side, the back, the shoulders, and even the right arm, and much more rarely down into the hypogastrium and the thighs. In some paroxysms the pain increases to extreme severity; and convulsive movements have been repeatedly observed, particularly in nervous persons, as a result of the pain. Some patients assume the most peculiar positions, such as doubling themselves up to dull the pain. The pain may be accompanied by nausea, eructations, and repeated vomiting. The vomitus usually consists of strongly bile-colored fluid and food remnants. The bowels are usually constipated, but sometimes there is diarrhea. The constitutional disturbance is marked; the patient feels extremely feeble and exhausted, and gives the impression of anguish and collapse. The pulse is small, usually accelerated, but sometimes infrequent. The bodily temperature is normal in a part of the cases; often there is slight fever, and sometimes a considerable rise of temperature, to 104° F. (40° C.) and more. At the beginning of the attack, and also later, there is apt to be chilliness, or a well-marked rigor; and this is often, but not invariably, associated with high fever.

Upon objective examination of the abdomen we find its upper portion, particularly the region of the liver and gall bladder, slightly prominent or even markedly so. The same region is sensitive to pressure, and this tenderness may be diffuse or localized. Sometimes the point of greatest sensitiveness is in the back to the right of the lower dorsal vertebræ (Boas). Sometimes an enlargement of the liver can be distinctly made out (*vide infra*), and sometimes when the abdominal walls are yielding we can, upon careful palpation, feel the enlarged, tense, and tender gall bladder (*vide infra*). In exceptional cases, it is claimed that we can even feel the grating of the stones upon one another, and with a stethoscope hear the crepitation thus produced; but in other cases it is impossible to make out with certainty any change, either in the liver or the gall bladder; or the abdominal walls may be so thick and rigid that a good examination is impossible. In any case of this sort the physician looks with special attention for the appearance of jaundice. Even a slight icteric hue of the eyes and the skin is important, for the appearance of jaundice is often decisive in leading to an absolute diagnosis. Sometimes the jaundice is very marked; in such cases the urine contains an abundance of bile pigment. It cannot, however, be too much insisted upon that it is by no means exceptional to have no jaundice at all in an attack of biliary colic (*vide infra*). Not very rarely, the icteric discoloration of the skin is absent,

while the examination of the urine distinctly shows some bile pigment. Itching of the skin is quite frequent with or without marked jaundice. The stools in continuous biliary obstruction become light, acholic, and fatty. With regard to finding gallstones in the feces, *vide infra.*

The duration of an attack of biliary colic varies greatly. Many attacks last merely a few hours or a day; more severe attacks continue two to four days, and even much longer. It is equally impossible to establish any universal rule as to the frequency with which the attacks recur. Sometimes a second attack will come after a short interval; sometimes not until months or years have elapsed. Not infrequently there will be several attacks in close succession, and then none for a long time; or there may never be any more. Again, the early attacks of simple colic may be followed later by a complicated disorder, due to one of the numerous sequelæ to which gallstones may lead.

We intend to discuss the remote results of gallstones, but we must first answer the question: What abdominal processes occasion the attacks of biliary colic, of which we have just presented the clinical picture? Until within a few years the answer to this question seemed very simple. Almost all authorities conceived that the attack was due mainly to the moving about of the calculi and their expulsion. It was said that the stones moved into the cystic duct because of their own weight, the pressure of the diaphragm, and, above all, the contractions of the gall bladder; and being once in the duct, they were forced along through the cystic and the hepatic ducts. The attack was successfully ended when at last the concretion escaped from the common duct into the duodenum. The incarceration of the stone in the common duct necessitated jaundice. If the stone stopped in the cystic duct, or, as was often supposed to be the case, went back into the gall bladder again, there would be no jaundice.

This old doctrine of the causes of biliary colic has lately been modified and extended in many important points. This has been particularly the result of the experiences of various surgeons (Riedel, Kehr, Körte, and others) in the operative opening of the stone-filled gall bladder and the biliary passages. Even now it must be confessed that small stones can be driven from the gall bladder through the cystic and common ducts into the intestine in the way above mentioned. If the stone is very small, its passage may take place almost without symptoms, and produce only slight signs which can scarcely be definitely interpreted. The passage of somewhat larger stones, however, produces typical attacks of biliary colic in the older sense. In our opinion many comparatively brief attacks, often repeated, very painful, and accompanied by vomiting and slight jaundice, can scarcely be otherwise interpreted. We believe that the chief cause of the pain are the cramp-like contractions of the smooth muscle fibers. In other cases, besides the mechanical obstruction of the stone, there is an inflammatory process, or the inflammation may occur primarily without any previous impaction of the calculus. Riedel depicts these processes, which take place with particular frequency as follows: So long as the stones in the gall bladder remain there without exciting any secondary change, and so long as the bile flows freely around them, in and out, there are no clinical symptoms, although there is very often a gradual development of what is called dropsy of the gall bladder. If a stone enters the neck of the bladder and there increases in size, or if the cystic duct becomes obstructed by

the swelling of its membrane, then the entrance of the bile into the gall bladder is impeded. The constituents of the bile which is in it are absorbed, and the contents of the gall bladder change to a yellowish serum, which may finally be almost colorless. This process often goes on without any symptoms, but frequently there are slight suggestions of chronic inflammation. The wall of the gall bladder becomes thickened; there may be a gradual formation of manifold adhesions between the serous coat of the gall bladder and the neighboring parts, such as the omentum, duodenum, or transverse colon; and these adhesions are in many cases the cause of the mild and indefinite pains and other unpleasant sensations of which many patients with gallstones complain for a long time previous to their first genuine attack. When a gall bladder has been changed in this way, there is liability to frequent and sudden acute inflammatory symptoms; acute calculous cholecystitis is the true anatomical basis of biliary colic. These inflammatory gallstone attacks are sometimes mild and of short duration, but, as a rule, they last longer than the attacks due to impaction alone. They may last several days, and are often associated with elevations of temperature and signs of peritoneal irritation. The exciting cause of these acute inflammations, which often recur or show exacerbations, is probably some infectious agent derived from the intestine (*Bacterium coli*), or occasionally, perhaps, from the tonsils or from some other place, and entering the gall bladder by way of the blood (diplococci, streptococci). Frequently, in these attacks, there is no migration and expulsion of the calculi into the intestine, and Riedel classifies them as "unsuccessful gallstone colic." They are, of course, as a rule, not associated with jaundice. If, however, the stone, situated at the neck of the gall bladder, is small and the cystic duct patent, the stone is pushed on by the pressure of the inflammatory exudation which collects behind it. We have now the pain due to the inflammation and also that due to the incarceration of the stone, and as soon as the stone reaches the common duct, and for a time blocks the discharge of bile, we have obstructive jaundice. If the stone is successfully driven through the common duct into the duodenum, the pain and inflammation usually promptly cease. By careful search we may find one or more stones in the feces. In brief, we have to deal with a so-called "successful" attack of biliary colic. The distinction between unsuccessful and successful attacks is, of course, not always self-evident; but it should be noted that, according to Riedel, unsuccessful attacks are much more frequent than the successful. It is true that in the successful attacks there is always jaundice, and in the unsuccessful attacks there is usually no jaundice, yet this criterion is not absolute, because, as Riedel was the first to point out, jaundice may occasionally be present in the unsuccessful attacks. In such a case the jaundice, of course, is not due to closure of the common duct by a stone—that is, it is not lithogenous, but it is inflammatory. The catarrhal inflammation of the gall bladder may sometimes extend to the bile ducts and produce jaundice, because of inflammatory swelling of the common or hepatic duct.

These mechanical and reflex processes and this simple cholecystitis or cholangitis (inflammation of the bile ducts) do not by any means comprise all of the pathology of cholelithiasis. In many cases there is not a simple cholecystitis or cholangitis, but a purulent inflammation, which is either purulent from the start or a sequel to a serous exudation. That bacteria are factors

in this process there can be no doubt. The condition now presented is no longer a simple attack of colic, but a far more severe and persistent constitutional disturbance, associated with pain in the hepatic region, vomiting, possibly jaundice, and very likely signs of enlargement of the gall bladder and the liver. There are high fever (often accompanied with rigors), general prostration, and cerebral symptoms. In milder cases there is only a purulent cholecystitis. In the severer cases there is a purulent diffuse cholangitis alone, or combined with purulent inflammation of the gall bladder. To enumerate here all the possibilities of the condition is impossible. Even severe derangement of this sort is not incompatible with spontaneous recovery, but in some cases, if it is too late for surgical aid, there is general septic infection and death.

A number of other sequelæ of gallstones may develop as a result of the impaction of the stone in the bile ducts, be it in the cystic or in the common duct. If the stone lodges in the cystic duct, there usually develops a large, often pear-shaped and palpable tumor of the gall bladder, the so-called dropsy of the gall bladder. If the stone lodges in the common duct there is usually long-continued jaundice with biliary congestion in the liver, which may finally lead to the so-called secondary biliary cirrhosis of the liver. As a result of secondary enlargement of the common bile duct, slight changes in position of the stone, etc., the passage of bile may again become free for a longer or a shorter time. This explains the frequent variations in the intensity of the jaundice despite a continuous impaction of the calculus in the common duct. Quite frequently, even without actual suppuration, an intermittent fever develops as a result of secondary infection (*fièvre hépatique intermittante*). We may only assume the presence of advanced suppuration when severe general septic symptoms are present. These occur especially when there is a considerable pressure necrosis of the surrounding tissue. This immediately opens the door to inflammatory germs, and results in secondary suppuration, which may be either diffuse or localized. In the worst cases there may be purulent phlebitis of an abdominal vein, with metastatic abscesses in other organs. Those cases without actual perforation occasion merely chronic inflammatory adhesions and contractions in the vicinity of the bile duct.

Precisely similar consequences may, however, arise from stones which are situated in the gall bladder itself (usually at its neck), with a consequent suppurative infection of the neighboring parts. Perforation into the abdominal cavity leads to purulent peritonitis. If adhesions have already been formed, as is usually the case, there may be perforation in any one of many directions, either outward with escape of the pus and often the gallstones, and perhaps with the formation of a biliary fistula, or into the transverse colon, or more rarely into the stomach, the portal vein, or the urinary tract; but chief in relative frequency and importance is perforation from the neck of the gall bladder into the duodenum, for this, as Virchow and Fiedler have pointed out, is the most common way by which large gallstones reach the intestine and come to be discharged with the feces. Many of these processes may, under favorable circumstances, lead to spontaneous recovery from cholelithiasis. The gall bladder, under the influence of suppuration, may finally completely shrivel up, perhaps retaining in its folds one or more calculi; but much oftener, if there is no prompt surgical intervention, the termination is unfavorable. The

associated clinical conditions are manifold and they cannot be sketched in detail, and the end may come in an acute and stormy manner, or after a long illness marked by many fluctuations. Still, we feel bound to emphasize, in closing, that the frequency of these many grave results of cholelithiasis which we have mentioned must not be overestimated. In comparison with the extremely great number of gallstones which produce either no symptoms at all, or none which suggest danger to life, the severe cases of purulent inflammation after cholelithiasis are few. Another severe, but fortunately likewise very rare, sequel of large gallstones is intestinal obstruction from impaction of the stone in the intestine. This has already been referred to (page 615).

Diagnosis.—It is evident from what has been already said that often the diagnosis of cholelithiasis is easy and indubitable, while in other cases the symptoms and course of the disease are obscure and ambiguous. The attacks of colic are certainly the most characteristic symptom. We should therefore make it a rule, in case of severe paroxysmal pain in the region of the stomach or liver, to think of the possibility of gallstones, and to try to discover by a most careful inquiry if the pains can be explained by a cholelithiasis. The aphorism of Pel applies, above all, to gallstones: "*Qui bene interrogat bene diagnoscit!*" The main points to be elicited are the sudden onset of the pain in the later afternoon hours or at night without particular cause, their great severity, their relatively short duration, their association with vomiting and chills, and their radiation to the back and shoulders. Furthermore, we must investigate the occurrence of previous attacks, a possible hereditary predisposition, and the presence of special ætiological factors, such as pregnancy [typhoid fever], etc. If an attack of this sort is associated in even a slight degree with jaundice, the diagnosis is usually perfectly clear. If there is no jaundice—and frequently there is none—the diagnosis is less certain, but still it can often enough be correctly suspected because of the general character of the painful attack, and the subsequent period of complete freedom from pain, as distinguished from the pains of gastric ulcer.

In our objective examination we must first look for local tenderness in the region of the gall bladder, and then, above all, the existence of a palpable tumor of the gall bladder or an enlargement of the liver. In examining it is best to place the left hand behind the right lumbar region of the patient, and by pressure to try to push the liver forward while deep palpation is carefully made with the right hand. The gall bladder often only becomes palpable on deep inspiration. The liver itself may be unchanged or enlarged on account of biliary obstruction or inflammatory swelling. As has been mentioned, a corset liver is often found in cholelithiasis. Occasionally that portion of the liver which is over the gall bladder is unusually enlarged downward (so-called Riedel's lobe). Sometimes the neck of the gall bladder is considerably elongated, so that the gall bladder itself forms an elongated, freely movable tumor, that may easily be confused with a movable kidney, or even with a movable carcinoma of the pylorus or intestine.

After we have decided that an existing attack of pain is the result of gallstone disease, we must then decide further whether we have to deal with a simple colic or with an inflammatory complication, such as cholecystitis, cholangitis, or pericystitis. In this regard the most important differential points are the duration of the attack, the presence of fever, the local rigidity

of the abdominal walls [and the ribs], and the sensitiveness to pressure, the general condition, the vomiting, etc.

In a certain number of cases, to be sure, it will not be possible to get beyond a mere suspicion that there are gallstones, and occasionally it will not be entirely possible to avoid confusion with intestinal colic, renal colic, pancreatic colic, the pains of a movable kidney, and purely nervous visceral neuralgia; yes, even with arteriosclerotic and anginose attacks, and with the gastric crises of tabes. The diagnosis is also particularly difficult when the attack is not a well-developed one with characteristic symptoms, but is more indefinite and suggests cardialgia, dyspepsia, and similar conditions. A careful and thorough examination of all the organs that might be involved, such as the stomach and kidneys, may in many cases aid us; and often the truth is revealed by the further course of the disease, but many cases remain obscure. The hope that gallstones might be recognized during life by means of the Röntgen rays has only been fulfilled in isolated instances.

We must say a word about finding the gallstones in the feces. Of course the discovery of the *corpus delicti* in the evacuations after an attack of colic makes the diagnosis absolute. They are to be searched for by mixing the stools with water, and then passing them through a sieve. But, as we have pointed out above, the "unsuccessful" attacks of biliary colic are probably much more frequent than the "successful." Perhaps some gallstones may also become disorganized in the intestine. We cannot, therefore, be surprised that in many cases of cholelithiasis no stones can be found in the dejecta, and Riedel is right in maintaining that it is not worth while to search for the stones except in those attacks which are associated with jaundice—that is, those that are not unlikely to be "successful." It should be pointed out further that we should be on our guard not to mistake for gallstones hardened portions of feces—for instance, compacted vegetable matter.

Often the diagnosis is very difficult in cases of severe and long-continued purulent inflammation, originating from gallstones. If there are such symptoms as indicate a peritonitis localized in the hepatic region, associated with fever and rigors, and suggesting circumscribed suppuration, we must always consider the possibility of gallstones being the primary cause. We should make a careful local examination and consider the general conditions, such as age and sex, and we shall often be greatly helped by inquiring carefully into the previous health of the patient—e. g., with regard to any characteristic colic. It must be confessed that it will not always be possible to avoid confusion between this and other suppurative processes, particularly those originating from old gastric and duodenal ulcers, from an abnormally situated appendix, or from suppurating echinococci. No special diagnostic rules can be formulated, for the symptoms are peculiar in almost every single case. Even when we are able to make a correct diagnosis of cholelithiasis we are often unable to estimate the exact condition, the extent of the secondary inflammation, the number and the situation of the stones, or the presence of secondary adhesions.

Prognosis.—The prognosis of gallstones must really be termed doubtful in every case, that is, if we provisionally exclude the possibility of cure by surgical means. The presence of gallstones, as the reader already knows, may entail a number of dangerous sequelæ, and we can never foretell whether the

patient will suffer from these or escape them; still, there is no doubt that in some cases, when the stones are small, or after perforation and emptying of the gall bladder into the small intestine, there may be a spontaneous and permanent recovery, while in other cases there may be disturbances of the most varied kind, persistent, or constantly recurrent, and yet never absolutely serious. There is no occasion for describing here a second time the numberless possibilities of cholelithiasis and their prognostic significance.

There is one important symptom which is interesting from the standpoint of general pathology and which must be noted here—viz., the development, subsequent to cholelithiasis, of cancer of the gall bladder and bile ducts. This possibility is, of course, of great clinical importance, and agrees with the general observation that persistent mechanical irritation of the mucous membrane and scar formations may occasion the development of carcinoma. The formation of secondary cancer after cholelithiasis is precisely analogous to the growth of cancer after ulcer of the stomach, or secondarily to stone in the kidney.

Treatment.—Our means of efficient prophylaxis against the formation of gallstones are very limited. The most we can assume is that an entirely suitable regimen, and particularly the avoidance of any tight lacing or similar mechanical interference with the excretion of bile, will prevent or at least delay the development of any tendency to the formation of calculi. If gallstones have been once formed, our task as physicians consists merely in moderating the disturbance which they produce; in guiding, so far as possible, all sequelæ to a favorable termination, and finally in effecting the removal of the stones from the body, so far as this is within our power.

If the symptoms of the patient remain comparatively slight, and especially if the diagnosis of gallstones has not yet been absolutely established, energetic treatment is not demanded. We regard it as important that in such cases, as soon as we conceive the possibility of cholelithiasis, the patients should enter upon a proper rest cure. Very often female patients of this sort are regarded as nervous, are sent into all sorts of health resorts, and treated with electricity and massage, and, of course, without success. Instead we should induce such a patient to spend a few weeks of complete rest in bed, or, at any rate, lying on a sofa with easy clothing; prescribe a cautious and nourishing diet, and the regular application of warm poultices upon the hepatic region, and administer before breakfast and in the middle of the forenoon a half pint of warm Carlsbad Mühlbrunnen water. We may then hope for a satisfactory improvement in the symptoms due, in our judgment, to the cessation of the slight inflammatory irritation. Often patients of this sort are sent to Carlsbad. We will not deny that Carlsbad water may have some favorable influence upon cholelithiasis, but we believe that the exertion of the journey and the promenades and other activities in which the patients are apt to indulge after their arrival, are often serious drawbacks. That Carlsbad water, as such, is of real value in expelling the stones or in actually dissolving (!) them, we find it difficult to believe.

Whenever there is a well-marked attack of biliary colic we have special indications for treatment. Of course, in this case also, we must immediately enjoin complete rest in bed.

The pain demands the prompt employment of narcotics, particularly of

morphin or opium. If the suffering is extreme, a subcutaneous injection of morphin is by far the best and quickest remedy, but the internal use of opium is also to be recommended. We may give every two or three hours 15 or 20 drops of the tincture, or 0.5 to 1 gr. of powdered opium (gm. 0.03 to 0.05).[1] The use of opium suppositories or small enemata (20 to 30 drops of tincture of opium in 5 drachms [gm. 20] of tepid water) is often very effective. Other narcotics, such as chloral or belladonna, are scarcely ever necessary, but we may sometimes combine extract of belladonna, which is much praised by many physicians, with the opium. Of external applications upon the hepatic region the best is a warm or hot poultice. It is exceptional for the patient to prefer an ice bag. Usually gentle rubbing of the hepatic region with chloroform oil (a mixture of equal parts of chloroform and olive oil) does good. In a few cases the patient experiences relief from a prolonged warm bath. If there is violent vomiting we give tincture of opium, bromid of potassium, cocain, or bits of ice. If there is marked collapse, such stimulants must be employed as wine, strong black coffee, or even injections of camphor. When the acute symptoms have ceased, we must still persist for some time in a cautious diet and in bodily quiet, and it is often well to prescribe a gentle laxative, such as Carlsbad salts or rhubarb.

When the attack is completely over, the question arises how to guard against the occurrence of others. The answer to this question depends mainly upon the view we take of the present attack, whether we regard it as a "successful" or an "unsuccessful" one. If the attack was successful—that is, if calculi were found in the intestinal evacuations after it—we may hope that in case there are more stones present they also may be discharged. How far we are able to hasten this process by artificial measures is a difficult question. We must adopt here, as is so frequently the case, the purely empirical standpoint. Under this heading, a course of waters at Carlsbad or similar watering places (Homburg, Ems, Tarasp, Mergentheim, Neuenahr, Bertrich, Vichy, etc.) must be given primary consideration. If the patient cannot visit a watering place, a methodical course may be prescribed at home. Thus, 0.5 to 1 pint of warmed Carlsbad Mühlbrunnen water may be slowly taken every morning on an empty stomach and also every afternoon.

Of the other remedies recommended for cholelithiasis, we would mention turpentine, formerly frequently used, but now only rarely. This may be used in the form of what is called Durande's remedy; that is, a mixture of ether and turpentine in the proportion of 3 to 2, of which two or three times a day 20 to 30 drops are given; salicylate of sodium, of which we have often given, per rectum, daily 0.5 to 1 drachm (gm. 2 to 4), in combination with bicarbonate of sodium. Finally, a number of drugs have been recently manufactured and frequently used. Among these are chologen (a mixture of podophyllin and calomel), cholelysin (oleate of sodium), and probilin. The administration of large doses of olive oil has been repeatedly recommended. Three to seven ounces (gm. 100 to 200) are given in divided doses daily with

[1] [When giving repeated doses of opium for biliary or renal colic, it should be remembered that the pain will cease abruptly as soon as the stone ceases to obstruct the passage, and that severe toxic effects of the drug may then appear. In bilious colic the distance to be traversed by the stone is relatively short, and in that affection, especially, the inhalation of ether of chloroform is sometimes the best, and, indeed, the imperative treatment.]

lemon juice. [Most of the oil is passed in lumps, which have a superficial resemblance to gallstones, and are sometimes called such.] All of these remedies appear at times to have a certain virtue, perhaps because they usually act as cathartics. They are, however, of no great value.

In all cases where the attacks occur very frequently and are associated with such severe symptoms that the patients are constantly miserable, we are justified in advising operation. In recent times the surgical treatment of cholelithiasis has achieved many brilliant cures. If one considers the possible dangers of the disease itself, it is easy to understand why some surgeons almost always recommend operative interference whenever the presence of gallstones has been demonstrated. Still, we must consider that the circumstances in practice may prevail over purely theoretical considerations, however lucid the latter may be. Many patients will not consent to an operation after a single brief attack, which, although painful, has ended in apparently complete recovery, and the conscientious physician will consider in any actual case the indisputable dangers of the operation and the possible doubt as to diagnosis. I am very loath to advise operation to patients with severe chronic jaundice, as I have often seen an unfavorable outcome in such cases as the result of uncontrollable hemorrhage (hemorrhagic diathesis)!

We must therefore carefully consider the surgical indications in each individual case, and in all mild cases first try to improve the condition of the patient by other means, such as a rest cure, a course of waters at Carlsbad, etc. If no result is obtained by these palliative measures, then the question of operation must naturally be given closer attention. An operation is necessary when the symptoms indicate a purulent cholecystitis or a localized abscess, due to gallstones. It would probably be better if such complications of cholelithiasis were avoided by anticipatory surgical interference, but this desirable result will never be attained in practice, because in many cases the purulent infection of the gall bladder or the bile ducts occurs without having been preceded by any marked symptoms. In circumscribed suppurations, surgical intervention often not only saves life but works a permanent cure, unless the purulent infection has already extended to the bile ducts of the liver itself, or has led to general pyæmia. We cannot in this place enter into all the particulars of the surgical treatment of gallstones or the various operative methods. Complete information will be found in surgical monographs.

CHAPTER III

SUPPURATIVE HEPATITIS

(Hepatic Abscess)

Ætiology.—Exclusive of traumatism there are two ways by which bacteria may penetrate into the liver, there to excite a suppurative inflammation—namely, by the blood and through the bile ducts. In the circulatory system the main route is by way of the portal vein, by which pyogenic organisms from the intestines reach the liver. This explains why many ulcerative processes in the intestines, such as severe dysentery, are followed by hepatic abscess, and why

other suppurative processes within the portal system, particularly the suppurative thrombophlebitis after appendicular inflammations (*q. v.*) may have a similar sequel. In general pyæmia the germs must take a very circuitous route in order to reach the liver. They must, on leaving the primary abscesses, first enter the veins and the lungs, and then gain the liver by way of the hepatic artery. It has been well known for a long time that suppurating wounds of the head are followed by hepatic abscess with comparative frequency. We have observed fatal suppurative hepatitis subsequent to a fetid bronchitis. Perhaps it may exceptionally happen that infectious matter enters the hepatic veins by "retrogressive embolism" from the vena cava.

The germs which make their way into the liver from the bile ducts invariably originate in the intestine. In these cases the hepatic inflammation is almost always preceded by disease of the biliary passages. The most frequent cause by far of this variety of hepatic abscess is the formation of gallstones in the gall bladder and in the liver. This has been fully discussed in the preceding chapter.

Among us, hepatic abscesses are rarely occasioned in other ways than those indicated; but in the tropics it is said that quite a large number of apparently primary hepatic abscesses are met with. Their origin is likewise to be attributed to the entrance of infectious material from the intestine. Most frequently, these liver abscesses develop after a tropical amœbic dysentery, and often after apparently mild cases. Bacillary dysentery, it must be mentioned, almost never produces a liver abscess. The amœbic abscesses are found chiefly in males, and much more rarely in those who are natives of the tropics than in those who have immigrated thereto.

Pathology.—The smallest and as yet imperfectly developed embolic abscesses best illustrate the mode of formation. We find the blood vessels choked with micrococci, and the cells of the surrounding parenchyma void of nuclei and in process of disintegration. Along the course of the blood vessels nuclei are very abundant. These are due to white corpuscles which have escaped through the vascular walls. The cells and the liquid exudation rapidly increase, and there is complete destruction of the hepatic parenchyma, and the formation of an abscess in its place. This extends in all directions. Large abscesses may at last involve an entire lobe. The tropical liver abscesses are situated almost without exception in the right lobe of the liver. In other cases the suppurative process is limited by encapsulation. Sometimes quite large portions of the liver become necrotic and slough off, under the influence of what is called "sequestrating" suppuration. We almost invariably find some shreds of hepatic tissue in the pus of hepatic abscesses. When hepatic abscess has been occasioned by gallstones, the suppuration is usually due to direct extension of the process from the bile ducts to the parenchyma of the liver. Not infrequently gallstones are found in the pus of such cases.

Small abscesses may perhaps be absorbed, but they are in most cases merely symptomatic of pyæmia or some such disease, which is itself incurable. Larger abscesses may point into neighboring organs. If they are discharged into the abdominal cavity, diffuse peritonitis follows. The most favorable termination, and one repeatedly observed, is perforation through the abdominal walls, after these walls and the liver have been joined by adhesions. They may also break

into the pleural cavity, the pericardium, the intestine, and the pelvis of the right kidney.

Clinical History.—An absolutely complete clinical description of hepatic abscess is impossible, because, as we have seen, it may be a symptom of such diverse pathological processes. Hepatic abscesses are often found postmortem which had given no previous indication of their presence; this is frequently true in pyæmia. In other cases there are symptoms, in part directly referable to the seat of inflammation, and in part due to its influence upon neighboring organs. In recent times, the pathology of tropical liver abscess has been particularly well elaborated.

Enlargement of the liver can almost always be made out by percussion and usually also by palpation. It is the result of swelling and hyperæmia involving the entire organ. Of particular importance is the percussion of the upper border of the liver to determine its height and the dome-shaped bulging of the right lobe. Extensive abscesses may give much more definite signs of their presence, however, if situated on the anterior surface of the organ. They are sometimes felt through the abdominal walls as hemispherical and actually fluctuating tumors. It is not so very rare for tropical hepatic abscess to attain these dimensions.

Pain in the right hypochondrium, although it may be entirely absent when the abscesses are small, even if they are numerous, is often violent and persistent when the abscess is large. It is excited by the tension of the peritoneal covering of the liver, or by perihepatitis. The pain often radiates—and with especial frequency into the neighborhood of the right shoulder. The exact determination of the tenderness on palpation is also important.

The course of the fever may prove a strong diagnostic point. When the abscess is chronic and encapsulated there may, it is true, be no fever whatever; but, as a rule, fever does exist, and it often presents a very characteristic intermittent character. There are great elevations, usually ushered in by a chill, and succeeded by deep depressions of temperature accompanied by perspiration. If the hepatic trouble is merely a symptom of general pyæmia, then the fever is to be ascribed to the latter; but if there are signs of a severe local hepatic disease, such as pain, enlargement, and perhaps jaundice, and if these febrile attacks come on at irregular intervals, we should always consider the possibility of abscess of the liver. In the cases of large tropical abscess this sort of fever is the rule. It is most frequent with us in cases of purulent pylephlebitis and of abscess excited by gallstones. The "*fièvre intermittente hépatique*" of the French is in most instances due to the presence of gallstones in the liver, with secondary suppuration and abscess formation.

Among the secondary symptoms of hepatic abscess jaundice is prominent. It is not invariably present, however, occurring only when the abscess has compressed some large biliary duct, and has thus given occasion to the absorption of bile by the lymphatics, or when an extensive cholangitis has simultaneously developed, sufficient to cause jaundice. In rare instances the abscess compresses the portal vein and thus causes ascites. There may be pulmonary symptoms of considerable importance, even when there are no actual pulmonary complications. This is because the right half of the diaphragm is crowded up by abscesses projecting from the convex surface of the liver. Compression of the cardia occasionally produce persistent difficulty in deglutition. Hiccough is

sometimes a source of distress, and it may be due to the pressure of the abscess upon the stomach. A distressing reflex cough may also occasionally occur. Vomiting is also a rather common and often very troublesome symptom. The occurrence of a leucocytosis is worth noting.

There is almost always great constitutional disturbance. The patient has no appetite, and loses flesh, particularly if there are frequent febrile exacerbations. Often there are severe nervous attacks. Very exceptionally, the disease remains latent for a long time, and does not disturb the general health to any great extent.

The course of the disease depends mainly upon the nature of the original disturbance. Severe pyæmic cases, in which hepatic abscesses develop, are generally brief, and are almost invariably fatal. Abscesses due to gallstones, and the large abscesses which are apparently idiopathic, are generally chronic, lasting for weeks, or even for many months. Cases exhibit manifold diversities, according to the position, size, number, and sequelæ of the abscesses. Among the possible results, we would once more call attention to perforation into neighboring organs. If the pus is discharged externally, recovery may ensue; as also if the pus reaches the intestinal canal or the bronchi, which seldom happens. Perforation into the abdominal cavity always excites a fatal acute peritonitis. As a general rule, hepatic abscess finally proves fatal, recovery being exceptional. Death is due either to the gradual loss of strength or to some complication.

Treatment.—Local bleeding, counterirritation, purgatives, and emetics are among the remedies which are advocated, but we can hardly expect them to exert much influence upon a hepatic abscess. The best way is to treat the case purely symptomatically, seeking to keep up the patient's strength and mitigate his suffering until, if we are very fortunate, we have a chance for operative interference. When once the other symptoms are reënforced by the discovery on palpation of a fluctuating tumor, the diagnosis is established, and the pus should be evacuated and the cavity drained. Particulars about the operation should be sought in works on surgery. More than one case of the large tropical abscess has been cured in this way; but the cases which are most common among us—namely, embolic abscesses and those excited by gallstones—hardly ever afford any opportunity for surgical interference.

CHAPTER IV

CIRRHOSIS OF THE LIVER

(Chronic Diffuse Interstitial Hepatitis. Laennec's Cirrhosis. Gin-drinker's Liver. Hobnailed Liver)

Ætiology and Pathology.—Cirrhosis of the liver is usually defined as a diffuse interstitial inflammation, chronic in duration, and resulting in a secondary atrophy of the true hepatic parenchyma. Weigert's careful study, however, of the analogous processes of "chronic interstitial nephritis" has shown that at least a large part of the changes which take place in the connective tissue are not primary, but secondary, and the consequence of a primary destruction of the genuine renal parenchyma. In our opinion we must adopt a

similar point of view for hepatic cirrhosis. We feel, therefore, that in most cases of hepatic cirrhosis the origin of the disease consists of a primary injury and consequent partial destruction of the hepatic cells, whereupon follow a secondary proliferation and final contraction of the interstitial tissue, just as is seen in lesions of the parenchyma of the kidneys, spinal cord, and heart.

Such a conception would be extremely compatible with one fact about the ætiology of the disease—namely, that chronic alcoholism is universally regarded as a potent predisposing cause. Hence the English name, "gin-drinker's liver." The harmful influence of alcohol can be appreciated if we remember that on being absorbed it is carried directly by the blood vessels through the portal vein to the liver. We assume that the poison exerts a specific injurious influence upon the hepatic cells proper, impairing their nutrition, and finally causing their destruction. That the disease attacks the periphery of the lobules and the interlobular connective tissue is easily intelligible. It is well known that the capillary anastomoses of the portal vein are situated between the lobules.

Hepatic cirrhosis is most common in those who drink distilled liquors, while excessive indulgence in less strongly alcoholic beverages, such as wine and beer, leads less frequently to cirrhosis. Still, we have repeatedly seen the disease in beer drinkers.

Until now, attempts to produce experimentally a hepatic cirrhosis in animals by chronic alcohol poisoning have given contradictory results. The interesting fact discovered by Bauer is, however, worthy of mention—viz., that after the administration of large quantities of alcohol in dogs, alcohol and also albumen (!) can be demonstrated in the bile.

The abuse of alcohol is surely the chief but by no means the only cause of cirrhosis. The disease sometimes attacks persons in whose case no such ætiology is possible. In such instances we are seldom able to demonstrate the real cause. The excessive use of spices, and other analogous substances, has sometimes been regarded as causative. It is also said that malaria and the acute infectious diseases sometimes leave behind them a tendency to cirrhosis. In our opinion such cases are due to the long-continued action of chemical agents, about which we do not as yet possess any accurate knowledge, which are conveyed from the intestine, or, perhaps, in some instances, also from the blood to the liver. These cases of "genuine hepatic cirrhosis" are analogous to the not infrequent cases of "genuine interstitial nephritis" of unknown origin. Perhaps in many instances, also, there is a constitutional and excessive sensitiveness of the hepatic cells to the injurious influences to which they are exposed while performing their normal functions.—The form of cirrhosis which follows diseases of the bile ducts (biliary cirrhosis as distinguished from portal hepatic cirrhosis), and also "syphilitic cirrhosis," will receive separate consideration. The liver changes in diabetes mellitus will be considered in the discussion of this disease.

Cirrhosis of the liver is seen much oftener in men than in women, and usually occurs in middle life. This is in harmony with the fact that the chief cause is the abuse of alcohol.

The anatomical changes are generally divided, without regard to the way in which they are brought about, into two stages. In the first stage the liver is uniformly enlarged, resistant, with its edge blunt and its surface at first perfectly smooth, but later presenting little dimples. On section, the increased consistence, or "interstitial induration" of the liver, can be readily perceived.

The acini are separated from one another by a relatively thick band of grayish-red interstitial tissue, and are at first readily distinguishable. Later on, the interstitial hyperplasia invades the acini themselves, and they cease to be discernible. If there is a marked fatty infiltration of the liver cells, the liver on section presents a distinct yellowish color. The microscope shows that the cause of this increase in size and firmness of the organ is due exclusively to the abundant cellular infiltration and the new formation of connective tissue between the individual lobules. Very frequently we find newly formed bile capillaries in the interstitial connective tissue. The neighboring cells of the parenchyma exhibit signs of disintegration, undergoing either simple atrophy or else fatty degeneration.

The second stage corresponds with the process of contraction of the newly formed connective tissue, but in this stage the destruction of the proper hepatic tissue has already assumed grave proportions. Under this process of contraction the liver undergoes progressive atrophy, and its surface becomes mammillated. The size of the nodules varies. The size of the whole organ may be reduced one half, or even more. Frequently its general contour is considerably modified. Often the diminution of the size of the liver is confined mainly to the left lobe. The serous coat of the liver is usually thickened and rendered opaque; the hepatic tissue feels very resistant and cuts with difficulty, often with a grating sound; the color of the cut surface is usually a rather light yellow (κιρρός = yellow). Upon microscopic examination, we find merely vestiges of parenchyma, embracing which are wide, firm bands of connective tissue. Even within the acini there is decided interstitial hyperplasia along the blood vessels. The hepatic cells exhibit various stages of fatty degeneration and simple atrophy. Brown masses of pigment are often found here and there, which have been left behind by the hepatic cells now destroyed. Regenerative changes can also be detected quite frequently. The most common of these is the formation of small biliary passages in the broad bands of interstitial tissue.

Although this division of hepatic cirrhosis into two successive "stages" is useful for a schematic understanding of the disease, it must be emphasized that such a division does not always correspond to the actual conditions. Perhaps, as in chronic nephritis (*q. v.*), it depends on the kind and intensity of the active harmful influence as to whether we shall find a large liver with a smooth surface (analogous to the kidney of diffuse nephritis) or a small liver with a granular surface (analogous to the secondarily contracted kidney). In many cases there seems to be no so-called "first stage" at all, and the previously normal liver is directly transformed into a contracted liver, perhaps comparable to the kidney of genuine interstitial nephritis.

Clinical History.—The onset of the disease is usually insidious. At autopsies quite an advanced stage of cirrhosis is sometimes found, to which not a single clinical symptom had pointed; and it is often observed that the duration of unambiguous symptoms is much shorter than the degree of anatomical change discovered postmortem would have led us to expect.

Cirrhotic disease of the liver may also be compensated for, for a long time, by the organism. In such cases we occasionally find, on examination of the liver, that it is distinctly enlarged and hardened without the patient showing any particular symptoms of the disease.

Very often, to be sure, certain prodromata appear long before the genuine

cirrhotic symptoms; but there is generally room for doubt whether these prodromata are excited by the incipient hepatic disease or whether they are not due to other coincident affections, such as the chronic gastric or intestinal catarrh which drunkards so often have. There are anorexia, nausea, epigastric uneasiness, eructations, constipation, and sometimes vomiting. There is evident constitutional disturbance in many cases, but in others the strength is unimpaired. The severer symptoms usually date from the time when disturbance of the portal circulation arises. We have already stated that the diseased process is most active in the interlobular connective tissue—that is, where the portal capillaries are situated. When the contraction of the connective tissue has resulted in the destruction of a large number of these portal capillaries and the minute veins from which they spring, the portal circulation is inevitably impeded, and there arises a passive congestion of the whole portal system. The signs of this are soon manifest.

The stasis in the veins of the peritoneum is, as a rule, the first to attract attention, from the ascites which it occasions. The distention of the abdomen and the sensation of weight and pressure due to this effusion are often the first things which excite the patient's attention and lead him to seek medical advice. Later on, the ascites sometimes becomes enormous, causing immense swelling and extreme tension of the abdominal walls, and, of course, proportionate discomfort. Proper nursing and internal treatment may diminish the ascitic effusion, and it occasionally disappears entirely. It quite often remains nearly uniform, until finally, for some reason, there is a change for the worse.

Next to ascites, the most important symptom of portal obstruction is enlargement of the spleen, which is due both to the increased amount of blood in the organ and to a diffuse hyperplasia of its tissues. As a rule, the increase in size is considerable, amounting sometimes to two or three times the normal dimensions. It is not very exceptional to find a well-marked splenic tumor in hepatic cirrhosis even before there is any ascites. In such cases the splenic tumor is not due at all to portal congestion but to other, perhaps toxic, influences, just as in biliary cirrhosis (*vide infra*). At any rate the discovery of splenic tumor is invariably of great diagnostic importance, although it is often no easy matter to make out the size of the spleen, inasmuch as both percussion and palpation are rendered very difficult by the presence of ascites. On the whole, palpation is the more reliable. Pain or other subjective symptoms are rarely observed. Exceptionally, there is no enlargement of the spleen. This may be due to the firmness and thickness of its capsule, or to the general marantic condition of the patient.

The venous congestion of the stomach and intestines excites catarrh, which is evinced by anorexia, nausea, and irregularity of the bowels. Usually there is quite obstinate constipation, but there may be persistent diarrhea. None of these symptoms occupies the foreground of the clinical picture, however, both because such symptoms are frequent in all grave chronic diseases and because many patients have had digestive derangements long before these severer troubles began. In less frequent but more important cases, the congestion in the gastric and intestinal mucous membranes reaches so extreme a degree that hemorrhages occur. If the hemorrhage is slight and comes from capillaries, there is diarrhea with blood-stained stools, or vomiting, the vomitus being

dark-colored. Probably the tendency to hemorrhage is aggravated by the general cachexia or by the alcoholism. Of more importance is the vomiting of large amounts of pure blood. This, as the author knows from repeated observations, may be sufficient of itself to endanger life. Such cases have more than once led to the incorrect diagnosis of gastric ulcer, but at autopsy we usually find well-marked varices in the œsophagus, due to passive congestion. The severe hemorrhage is occasioned by the bursting of a varix. We surmise that similar conditions, probably mostly in the rectum, cause the severe intestinal hemorrhages which are sometimes seen. Profuse surface hemorrhages by diapedesis may occur without varix or ulceration. These depend upon a form of hemorrhagic diathesis due to changes in the walls of the capillaries.

There is sometimes a slight jaundice in ordinary cirrhosis, which is due to the existence of duodenal catarrh, or perhaps to catarrh of the smaller bile ducts. In many cases, however, there is no jaundice at all, or scarcely any. Not infrequently then, however, the skin shows a peculiar dark, dirty-yellowish color, which is very characteristic of chronic hepatic disease.

The above signs of portal obstruction will often render the diagnosis of hepatic disease extremely probable, but we should always endeavor to confirm our opinion by physical examination of the liver. In the later stages of the disease, and particularly if there be great ascites, our efforts may be fruitless; but at first, or after paracentesis has been performed, percussion and palpation may yield valuable information. In the earliest stages the liver is usually large. Hepatic dullness reaches some way below the edge of the ribs, and we can often feel the lower edge and anterior surface of the organ. Later on we find the surface irregular and rough. If we can feel these little nodules or prominences through the abdominal walls, as we sometimes can, of course the diagnosis of cirrhosis of the liver is nearly certain. As already mentioned, it often happens that irregularities are already to be felt upon the surface of the organ while it still remains hypertrophic. The demonstration of atrophy by percussion in the later stages of the disease is less reliable. The ascites often interferes with such an attempt. We may also be misled by coils of intestine distended with gas and perhaps lying in front of the liver. If, however, after guarding against error, we constantly find the area of hepatic dullness diminished, the sign has some value. Moreover, it is not infrequently possible to make out by palpation that the liver, although reduced in size, is hard and uneven. This is easier after the removal of ascitic fluid, when the abdominal walls are lax, and also if the attachments of the liver have been relaxed so that it sinks downward. If the atrophied liver cannot be directly felt, I have at times been struck in palpation by the "emptiness of the right hypochondrium" —i. e., the possibility of reaching without resistance high up under the right costal arch.

General nutrition is usually much impaired in the later stages of the disease. At first the patient may retain vestiges of his former corpulence, but finally he grows emaciated. Anasarca may exceptionally occur toward the close, but there is frequently considerable œdema of the lower extremities, and even of the scrotum and the dependent portions of the abdominal walls. The cause of this is a purely local one—the pressure of the ascites impedes the return of blood from the lower limbs to the heart. Thus arises a condition which is characteristic of portal obstruction in general, and hepatic cirrhosis

in particular, and has been termed "œdema of the lower half of the body." There is ascites with œdema of the loins, the scrotum, and the lower extremities; while the upper extremities, the upper half of the trunk, and the face are entirely free from œdema.

Occasionally there are ecchymoses into the skin, the mucous membranes, the retina, and other parts. These are probably due to malnutrition of the vascular walls. The gastric and intestinal hemorrhages have been already discussed.

In uncomplicated cases of the disease there is no fever. The pulse, even when there are no cardiac complications, is small and often somewhat rapid. Respiration may be much embarrassed as a result of the upward pressure of the diaphragm.

At first the urine presents no characteristic changes. When the ascites has become considerable, and there is œdema, the urine grows scanty, dark, and concentrated, and often has an abundant sediment of urates. The frequently large amount of urobilin in the urine is worthy of mention. This is best demonstrated by the Schlesinger test—viz., fluorescence of the urine on addition of an equal amount of a ten-per-cent solution of zinc acetate in absolute alcohol. The demonstration of the presence of urobilin is not without significance in the diagnosis of hepatic cirrhosis (P. K. Pel), as we also can testify. An exact explanation of urobilinuria in liver disease is as yet not possible. It is generally assumed that the diseased liver cells are no longer able to dispose of the urobilin absorbed from the intestine. It must also be mentioned that some observers have found a diminished amount of urea in the urine, and have attempted to attribute this to a disturbance of the urea-forming function of the liver. More recent observations, however, have not confirmed this contention. Frequently, comparatively large quantities of ammonia have been found in the urine. This is probably due to an increased acid formation in the blood, and not to a diminution in the formation of urea. The statements of Bouchard relative to the increased toxicity of the urine in hepatic disease, in consequence of the diminished poison-neutralizing function of the liver, require further verification. Glycosuria and alimentary glycosuria occur occasionally, but by no means regularly. Worthy of note, too, is the assertion of H. Strauss and others, that levulosuria results more readily in patients with hepatic disease after the administration of levulose than it does in healthy persons. This also requires further investigation.

It remains for us to describe briefly the collateral circulation which may be developed in cirrhosis, so as to enable the blood of the portal system to reach the systemic veins. The clinical history of the disease does not indicate that this attempt at compensation is very successful. We may have: 1. Communications between the veins of the mesentery and of the abdominal walls. 2. Communications between the coronary vein of the stomach and the veins of Glisson's capsule on the one hand and the phrenic veins on the other. 3. Anastomoses between the internal hemorrhoidal and the hypogastric veins. 4. As pointed out by Baumgarten, enlargement of the not yet completely obliterated umbilical vein in the ligamentum teres. Through all these the blood may flow from the portal system into the veins of the abdominal walls —that is, in the reverse of the normal direction. Then in a few exceptional cases the veins around the navel become varicose, a phenomenon which has

been termed the "*caput Medusæ*." Much more frequent and more important is the finding of unnaturally distended and tortuous veins in the skin of the abdominal walls when there is ascites. These veins usually extend upward from the neighborhood of Poupart's ligament. This phenomenon, aside from possible anastomoses between the subcutaneous veins and the portal tributaries (*vide supra*), is due to the fact that because of the ascites the flow of venous blood from the lower extremities through the iliac veins is impeded, and consequently a part of the venous blood takes its course through the inferior and superior epigastric veins to the mammary veins.

The complications of hepatic cirrhosis which are seen in many cases are probably due in part to the same influences (alcoholism) which occasion the cirrhosis. In this list belong, for example, cardiac hypertrophy, intestinal nephritis, and chronic pachymeningitis. Of special interest, because of its diagnostic and therapeutic importance, is the combination of hepatic cirrhosis with tuberculous peritonitis. As we have already seen, hepatic cirrhosis is probably in most cases the primary disease, and occasions a predisposition to the development of tuberculous peritonitis. The clinical picture in these cases is a combination of the symptoms of hepatic cirrhosis, such as a palpable granular liver, splenic tumor, and yellowish complexion, with the symptoms of tuberculosis, such as irregular distention and tenderness of the abdomen, persistent fever, and emaciation. If we consider the ætiology (alcoholism), we may, therefore, sometimes make a complete diagnosis of this combination, particularly if at the same time the tubercular inflammation attacks other serous membranes, such as the pleura, or if there are other signs of a tubercular infection (lungs, etc.).

As to the general course of cirrhosis, its duration cannot easily be determined because the onset is usually insidious. As a rule, the disease lasts one to three years, or rarely longer. In many cases the symptoms are insignificant for the first six to eighteen months. Then the disorder takes on a severer form, perhaps rather suddenly. Ascites appears, for example. These graver symptoms persist, until after a few months the patient dies. The course of the disease reminds one of cardiac cases, where for a long while the compensatory changes avert any distress, until on a sudden the circulatory disturbances become pronounced and persist to the end. To be sure, in hepatic cirrhosis very marked symptoms (extreme ascites) may for a time completely disappear.

Prognosis.—The prognosis is always unfavorable, at least when the symptoms have once become well marked. It may be that in the earlier stages the disease can be checked or even permanently cured, but even this is open to doubt. No case recovers in which the diagnosis of cirrhosis is certain.

Death is due either to intercurrent disease, or more often to gradually increasing exhaustion. In some few cases severe cerebral symptoms suddenly appear: there are coma, general convulsions, and delirium, and these usually are soon fatal. Just how these nervous phenomena originate we do not certainly know (see the chapter on Acute Yellow Atrophy of the Liver).

Diagnosis.—The diagnosis of hepatic cirrhosis is not usually self-evident. It may be made with some positiveness if a patient who has a previous history of alcoholic excess gradually develops ascites and splenic tumor, and if upon palpation we find the liver unnaturally firm and hard, either enlarged or

shrunken, perhaps with a roughened surface, and in any case distinctly to be felt; yet in palpating the granular surface one is very liable to errors, and particularly the separate kernels of fat in the *panniculus adiposus* of the abdomen are apt to be mistaken by inexperienced observers for irregularities in the surface of the liver. The difficulties in the percussion of the liver have been already indicated. Often a patient does not come under observation until a considerable ascitic effusion has already taken place, so that physical examination of the liver and spleen is rendered very difficult, if not impossible. Then we must first exclude any general disturbance of circulation as a cause of the ascites. If the heart, lungs, and kidneys are found to be normal, and if there is no œdema in the upper half of the body, it is very probable that there is a local derangement of the portal circulation, particularly if the patient is positive that the abdominal enlargement was the first evidence of dropsy; but we have still to determine whether the cause of this derangement is cirrhosis of the liver. This may be assumed to be the case if the whole course of the disease warrants the assumption, and if the history furnishes that most frequent of all ætiological factors, chronic alcoholism. Otherwise we must be cautious, for portal obstruction with precisely similar results may be due to other causes—such as the external pressure of tumors or portal thrombosis. Many forms of hepatic syphilis (*q. v.*) cannot be differentiated from cirrhosis by mere clinical observation of the hepatic disorder. Here it is only the ætiology and the demonstration of other signs of syphilis that can justify the assumption that the disease in hand is of specific origin. It is often a great aid in diagnosis to examine the liver directly after tapping. The abdominal walls are then relaxed and soft, so that the liver may be made out very distinctly. It should be borne in mind that irregularities of considerable size are more frequent in hepatic syphilis ("lobulated liver"), while cirrhosis is suggested by a more uniform granulation.

It is also very difficult in many instances to exclude chronic peritonitis. The ætiology may aid us. Other points are, that in chronic peritonitis there may be tenderness on pressure, the abdominal distention is less uniform, and there is no enlargement of the spleen. Another point is that the specific gravity of a simple ascites due to passive congestion is often below 1.015, while an inflammatory exudation usually reaches higher figures. The albumen content of an inflammatory exudation is generally decidedly higher than that of a transudate. As a boundary figure, four per cent may be taken, though exceptions in both directions may occur. Finally, the Rivalt test deserves consideration (*vide supra,* page 355). Hemorrhagic ascites, as a rule, indicates tubercular or cancerous peritonitis. But even if an isolated transudate in the abdominal cavity has been demonstrated, a false assumption of hepatic disease not infrequently occurs, through the circumstance that occasionally in heart disease, particularly in obliteration of the pericardium and in mitral stenosis, an isolated ascites may develop. We have already discussed this question (*cf.* pages 402 and 448).

The combination of hepatic cirrhosis and chronic peritoneal tuberculosis (*vide supra*) may be diagnosticated with a certain probability if, besides symptoms of cirrhosis and peritoneal disturbance, there are definite indications of a tubercular affection; such are a phthisical constitution, persistent fever, and the existence of a tubercular disease in some other organ, particularly

the pleura. The lymphocyte content of the exudate is also of diagnostic importance (*cf.* page 361).

Treatment.—In treating hepatic cirrhosis the first necessity is a strict regulation of the diet. The earlier in the disease proper diagnostic rules are followed, the more likelihood of benefit. The most important principle in regulating the diet is to avoid all ingesta which in any way might irritate or damage the hepatic cells. We forbid, therefore, all alcoholic beverages, or at most allow very small amounts merely to stimulate the appetite. We likewise forbid sharp spices, pepper, mustard, horse-radish, onions, and similar articles. In general, the amount of meat should be limited. We may allow milk, buttermilk, eggs, vegetables, and cereals. In suitable cases we may try the prescription of an exclusive milk diet for some weeks. Quite a number of authors report favorable results from a milk cure of this sort, but the individual peculiarities of every case must be considered.

If the disease has already developed we must resign ourselves to merely symptomatic treatment. The benefit which iodid of potassium is said to exercise in hepatic cirrhosis is very doubtful, and is probably based merely upon cases of syphilitic disease of the liver.

The symptomatic treatment of hepatic cirrhosis has to deal mainly with the disturbances due to portal congestion, and the associated gastrointestinal catarrh. In such cases experience shows that the best results are obtained by the cautious use for a considerable time of laxatives. In early cases we should resort first to salines, particularly Carlsbad salts. We may prescribe artificial Carlsbad salts, dissolved in warm water, or the genuine Carlsbad water to be drunk at all times. If the patient's circumstances permit we may even recommend in suitable cases a visit to Carlsbad. Other appropriate health resorts are Marienbad, Kissingen, and Neuenahr. If the saline remedies are ill-borne we employ vegetable laxatives, such as rhubarb and aloes. In case there is already a considerable degree of ascites, good results may sometimes be obtained by administering drastic purgatives, among which gamboge has earned a special reputation. Many observers have seen benefit from calomel, giving three times a day 3 gr. (gm. 0.2) for a period of two to three days. Calomel may be given repeatedly in this way at intervals of from five to eight days. Of course, caution is necessary in its employment. Probably it does good both as a laxative and also as a diuretic. Sasaki, in Japan, has seen excellent results in hepatic cirrhosis from the administration of cream of tartar in considerable doses, 2 to 4 drachms (gm. 10 to 15) a day, associated with a nourishing diet, including milk and scraped raw meat. After all, however, laxatives should not be employed if they disturb the digestive apparatus.

If ascites has already developed, we may administer diuretics besides the drastic purgatives; trial may be made, first of all, with diuretin, theophyllin, caffein; further, with acetate of potassium or sodium, squills, the species diureticæ of the German Pharmacopœia, dog-rose tea, bean tea, etc. English authorities recommend the balsam and the resin of copaiba as particularly efficient in the various forms of ascites. The dose is about 15 minims (gm. 1) a day. The best mode of administration is in gelatin capsules. Pure urea (2.5 to 4 drachms [gm. 10 to 15] daily, dissolved in water) has also been recommended. We dare not place too much hope in all these remedies. If the heart is feeble, digitalis may cause a decided increase in the amount of urine,

and it is also worth while to try a combination of digitalis with calomel or with other diuretics.

If the ascites is so excessive as to occasion much local discomfort and to impede respiration, the removal of the fluid by paracentesis may afford relief. The details of this proceeding were given in the last section. Many physicians recommend tapping as early as possible, before it is absolutely necessary. The relief is said to be more lasting in that case; but this proposal has by no means met with universal assent, and, as a rule, the physician will not tap until the abdominal tension, the dyspnœa, and other symptoms occasioned by the ascites render it necessary. Unfortunately, the relief is only temporary, for the fluid collects again with great rapidity after tapping; yet it is sometimes possible to delay the reaccumulation for a considerable time by compressing the abdomen with an elastic bandage, and by using the above-mentioned remedies (diuretics and digitalis) directly after tapping.

We have finally to consider the ingenious suggestion of Talma to assist in the establishment of a collateral circulation between the portal tributaries and the veins of the abdominal wall, by suturing the omentum and the spleen, also, if necessary, to the anterior abdominal wall. This Talma-Drummond operation has already been frequently carried out in hepatic cirrhosis as well as in other diseases of the liver with portal obstruction (syphilis of the liver, portal thrombosis, etc.). Sometimes the result has been apparently satisfactory, inasmuch as after the operation the ascites did not return immediately and the patients felt much improved. In suitable cases, therefore, we may advise an operative procedure of this kind.

CHAPTER V

BILIARY CIRRHOSIS AND HYPERTROPHIC CIRRHOSIS OF THE LIVER

There are two forms of cirrhosis which differ in many respects from the disease just described: they are called biliary cirrhosis and hypertrophic cirrhosis of the liver. Charcot and Hanot were the first to call attention to them. Since then the literature of the subject has become quite extensive, but all doubts and differences of opinion are not yet settled.

1. SECONDARY BILIARY CIRRHOSIS OF THE LIVER

Whenever there is retention of bile in the liver for any length of time, no matter what causes it, certain changes result. The small and the medium-sized bile ducts become distended, and granules of pigment are deposited, both in the interlobular connective tissue and within the acini themselves. Besides this, however, and undoubtedly because of the noxious influence of the retained bile, the hepatic cells not infrequently undergo destructive changes. In accordance with the general rule, connective tissue gradually fills the gaps thus left in the parenchyma, and, more than this, the interstitial hyperplasia is so great as to increase the size of the organ. If, therefore, there is persistent obstruction of the common duct by a gallstone, or a cicatricial stenosis, or a tumor pressing from without upon the duct, the liver will, in all such cases, be

found to be larger, firmer, and richer in fibrous tissue than normal—in a word, "cirrhotic." Hence this condition does not represent an independent disease, but is a result of chronic biliary retention, in whatever way occasioned. It is appropriately termed "secondary biliary cirrhosis." That retention is really the cause of this change has been proved by experiments, for it has been shown that ligature of the common duct in animals causes well-marked biliary cirrhosis.

2. PRIMARY HYPERTROPHIC OR BILIARY CIRRHOSIS

The secondary cirrhosis just described is due to occlusion of the large bile ducts. There is also a not very common (at least in Germany, though more frequent in Holland) primary form of biliary cirrhosis, usually known as hypertrophic cirrhosis. French authors have given it the name of *"cirrhose hypertrophique sans ascite avec ictère,"* out of regard to its most important clinical symptom. That there is an essential difference between this form and the ordinary "atrophic" cirrhosis of Laennec is manifested by the clinical behavior of the disease.

The specific cause of hypertrophic biliary hepatic cirrhosis is as yet entirely unknown. We can merely surmise that we have to do with a chronic inflammatory process, excited by some toxic or infectious agent, and that the process probably originates in the small bile ducts of the liver. The disease occurs most frequently in younger individuals, between the ages of twenty and thirty. Sometimes this disorder attacks hard drinkers, but they are not by any means its only victims. While, in the common form of cirrhosis, ascites is usually the earliest grave symptom of disease, in hypertrophic cirrhosis a slight jaundice generally appears simultaneously with the first indefinite symptoms of pressure in the region of the liver, languor, and anorexia. This jaundice rapidly increases, and persists throughout the illness. It is worth mentioning that, despite the most extreme icterus and bile-colored urine, the stools in biliary cirrhosis are, as a rule, not entirely decolorized. In ordinary cirrhosis there may be, as we have said, hardly any jaundice, or, at any rate, it is a rather late symptom, and even then it is seldom extreme. On the other hand, ascites, as a rule, is slight or absent in hypertrophic cirrhosis. It is true that there have been cases with great ascitic effusion, but it never comes till the disease is quite far advanced.

On physical examination the liver is usually found to be considerably enlarged, and its surface is smooth, as a rule, or rarely it displays some low elevations, due, perhaps, to variations in the fullness of the blood vessels. In general there is said to be this important difference between the ordinary (atrophic) and the hypertrophic forms, that in the latter the newly formed connective tissue evinces little tendency to contraction, so that the liver remains large, even late in the course of the disease, and does not shrink. This is certainly true of the majority of cases, so that after an illness of two or three years the liver is still found to be large and smooth; but further observations are necessary in order to determine whether there may not sometimes be contraction toward the end of the disease.

Along with the chronic jaundice and the marked enlargement of the liver there is a third important symptom—viz., chronic splenic tumor, usually of

considerable size. As there are no indications of portal obstruction, we must conclude that the spleen is not enlarged from passive congestion, as is usually the case in ordinary alcoholic cirrhosis, but as the result of hyperplasia; and post-mortem examinations confirm this conclusion. The origin of the hyperplasia is not clear. In one case we found not only splenic tumor but a distinct hyperplasia of many mesenteric and peritoneal lymph-glands; which suggests the possibility of infectious influences. A corroborative fact is that not infrequently the entire course of the disease will be marked by slight elevations of temperature. Longer fever periods with intermittent higher elevations of temperature also occasionally occur. The other symptoms of the disease are in part the direct result of the jaundice, such as the slow pulse, the itching, and the changes in the urine; while others, such as languor and weakness, are the results of the constitutional disturbance. The dejections, as already mentioned, are not always colorless and fatty, as in case of obstruction of the large bile ducts. Many patients have a noticeable tendency to hemorrhage, particularly to frequent epistaxis, hemorrhage from the gums into the skin, and to hematemesis. In the case mentioned above, the fatal termination was due to constantly repeated and profuse hemorrhages from the stomach, for which the autopsy disclosed no anatomical cause in the stomach or intestine. It seemed as if the hemorrhages had taken place by diapedesis.

The entire duration of the disease is about two or three years; but it may last much longer. The prognosis is almost always bad. Occasionally a case will exhibit marked temporary improvement or an apparent arrest of the disease, so that, in spite of the size of the liver, the patient is able to do light work. Death comes, when there are none of the above-mentioned hemorrhages, as a result of gradual exhaustion, or is suddenly ushered in by coma, convulsions, and other grave nervous symptoms, usually ascribed to cholæmia (*vide infra*).

That a chronic hepatic disease of so peculiar a nature should be marked by peculiar anatomical changes would seem certain, even from clinical observation. In general, this view is confirmed, although further and careful investigations are much needed. Upon microscopic examination there is always found an abundant new growth of a tissue containing many cellular elements, surrounding the small bile ducts of the liver. In other particulars the histological changes are very similar to those of ordinary hepatic cirrhosis. The new growth of connective tissue is found in the interior of the hepatic lobules, but this can scarcely be regarded as a radical difference. Still, one gets the impression that the entire pathological process of hypertrophic cirrhosis originates in the bile ducts and the tissues surrounding them, while the changes of ordinary alcoholic hepatic cirrhosis start from the branches of the portal vein. The absence of contraction in the new tissue of hypertrophic cirrhosis seems to be consonant with the fact that the anatomical changes suggest hyperplasia rather than inflammation (compare the changes in the spleen and the lymph-glands).

The diagnosis of hypertrophic cirrhosis can sometimes be made with considerable positiveness, and sometimes can merely be regarded as probable. The gradual development and persistence of jaundice and the presence of an enlarged liver and spleen, but usually without marked ascites, would suggest the disease strongly, especially if long-continued observation enabled us to exclude

carcinoma and other diseases. Sometimes, however, it is very difficult to decide whether the biliary cirrhosis is primary or secondary, for the conditions giving rise to secondary biliary cirrhosis (occlusion of the bile ducts from scars, new growths, or calculi) may be obscure. A careful investigation into the history of the case with regard to biliary colic and similar symptoms might be decisive. Other factors are the general course of the disease and the presence of a splenic tumor, unaccompanied by ascites. An enlarged spleen is scarcely ever seen in this way in ordinary chronic jaundice of obstructive origin.

The prognosis of biliary cirrhosis of the liver is most unfavorable, although the duration of the disease may in some cases extend over a period of many years.

The treatment should conform to the principles laid down in the chapters on jaundice and ordinary cirrhosis of the liver. Sacharjin has lately recommended the persistent use of small doses of calomel—1 gr. (gm. 0.06) several times a day. I personally have never seen any distinct benefit from it.

CHAPTER VI

ACUTE YELLOW ATROPHY OF THE LIVER

Ætiology.—Acute fatty degeneration of the liver occurs both as a primary disease and as secondary to other hepatic disorders, or as a symptom of constitutional diseases. Secondary acute fatty degeneration in rare instances accompanies severe acute infectious diseases, such as typhoid fever, recurrent fever, septicæmia, and puerperal disease. It also appears, with equal rarity, in the course of cirrhosis of the liver or of persistent biliary retention, and it is a constant phenomenon in acute phosphorus poisoning. Indeed, the effects of phosphorus resemble the symptoms of primary acute yellow atrophy in many ways so closely, even postmortem, that formerly the two were often confounded.

Primary acute yellow atrophy of the liver is a very rare but extremely severe disease which almost invariably leads to speedy death. There is generally no determinable cause, and its victims are struck down in blooming health. It is most common in young adult life between the fifteenth and thirty-fifth year. Children and elderly people have been occasionally attacked. Females are much more liable to the disease than males, and pregnancy increases the predisposition to it.

As we have said, we cannot, as a rule, find any exciting cause. It is stated that sometimes the onset has been preceded by some violent emotional excitement, or excess in alcohol, or the like; but how important these factors may be is not at all clear.

It is an interesting fact that sometimes the disease becomes rather more frequent than usual, and endemic. For instance, several members of one family may be attacked. This fact, the whole course of the disease, and the pathological appearances, seem to place the disease in the category of acute infectious diseases. It must be confessed that as yet we know nothing about the intimate nature of the infection.

Pathology.—The chief change found postmortem is in the liver, and it has determined the name given to the disease.

The liver is much atrophied, sometimes being only one half or one third its normal size. This makes its capsule often seem contracted and wrinkled. The organ is usually soft and flabby, so that in some places it seems as if the finger could be pressed into it. The color of the surface, and for the most part of the cross-section also, is yellow, like ochre or saffron; but the cut surface may be particolored, having red and yellow spots interspersed. Hence the names "red atrophy" and "yellow atrophy." The arrangement and relative extent of these patches may vary exceedingly. The red places look as if they had collapsed, and seem tougher than the yellow. They correspond, as we shall soon see, to the more advanced stages of the affection, while the yellow spots have undergone less change. The lobules are, as a rule, no longer distinguishable by the naked eye. Such lobules as can still be made out seem abnormally small and have a gray periphery.

On microscopic examination, we find that the essential change is an intense and uniform fatty degeneration of the hepatic cells, affecting the entire parenchyma. Only a few cells still retain their normal condition. The others are filled with large and small fat globules, and many are already suffering evident disintegration and absorption. Where the degeneration is furthest advanced, fat, detritus, and pigment alone remain. Inasmuch as the lymphatics readily absorb and remove the fatty and albuminoid granules, there is finally little left except blood vessels and connective tissue. The blood vessels are frequently quite congested, and thus occasion that red color which the naked eye detects in the more advanced, broken-down portions. Frerichs made an interesting discovery, which deserves mention, of the existence of leucin and tyrosin crystals both in the parenchyma and in the blood vessels. Bilirubin crystals also are sometimes found in the detritus, and more rarely in the interior of the hepatic cells.

Not only the liver, but many other organs present fatty degeneration: the heart, in particular, the kidneys, and rarely the muscles; but the process is always most intense in the liver. The skin (*vide infra*) and most of the viscera are evidently tinged with jaundice.

Acute splenic tumor is invariably present. This suggests that the disease may be infectious. That the disease is a constitutional one is also to be inferred from the numerous ecchymoses in the skin and the interior of the body, especially in the mucous membrane of the stomach and intestines, in the serous membranes, in the pelvis of the kidneys and the kidneys themselves, and more rarely in the brain and heart. This, again, is like the grave septic diseases. The blood itself is dark, with few clots. Leucin and tyrosin have repeatedly been detected in it. The peritoneum and other serous cavities sometimes contain a considerable amount of serum.

Clinical History.—The disease is usually divided into two stages, the first of which corresponds to the milder prodromal symptoms, the second to those severe symptoms which are alone characteristic. In many instances, however, the first period is wanting, or is so brief that the patient is plunged almost without warning into the gravest condition.

The prodromata in most cases consist of constitutional disturbances and mild gastrointestinal symptoms. The patient is languid, without appetite,

and disinclined to exertion. There are headache, nausea, vomiting, slight pains in the gastrohepatic region, and sometimes moderate fever. In a few days jaundice usually appears. This is almost invariably taken for an ordinary catarrhal attack.

After some days, or it may be weeks, the second stage begins. The chief characteristic of this is the occurrence of grave nervous symptoms. First there is violent headache, with sleeplessness and marked restlessness. The intellect is usually somewhat dulled even now, and articulation is slow and clumsy. The mental confusion usually advances very rapidly to a noisy and violent delirium. The excitement becomes at times maniacal. The patient is very restless, and he can hardly be kept in bed. Often there are convulsive twitchings of individual muscles, and there may be typical epileptiform attacks, but this is not common. After one or two days, or rarely longer, the excitement abates, and is followed by sopor, which soon passes into deep coma. At death the patient is usually perfectly unconscious. It is exceptional for the excited stage to be wanting; in such cases the first nervous symptom is sopor.

The cause of the nervous symptoms has not been explained in a way to silence discussion. The same theories which have been set up to account for the grave form of jaundice in general (see Appendix to this chapter) have also been employed to elucidate the nervous phenomena of acute yellow atrophy. It might also be that the primary specific processes of infection or intoxication are factors.

The jaundice, which is present even in the first stage, afterwards usually deepens. The urine contains bile pigment, and many investigators have also found bile acids in it. If these latter are present, it suggests that the jaundice is not (as was formerly believed) hematogenous—that is, the result of a destruction of red blood corpuscles and the transformation of their pigment into biliary coloring matter—but is due rather to a retention of bile. Just how this retention arises we do not yet know definitely. The obstruction cannot be in the large bile ducts, for the gall bladder is usually found empty. Hence the cause of the retention of bile and of the jaundice is probably a derangement of the smaller biliary passages within the liver. We should add that in a few rare cases there has been little or no jaundice.

On physical examination of the liver during the last stage of the disease there is usually a striking diminution of hepatic dullness, corresponding to the atrophy of the organ. Generally the first change to be detected is a contraction of the left lobe, as shown by the development of tympanitic resonance in the epigastrium. At the commencement of the illness the hepatic dullness is normal or slightly increased in area. If the disease proves very rapidly fatal the organ may never become very small. In most cases, though by no means in all, there are pain and tenderness in the hepatic region, but these are seldom so great as in phosphorous poisoning.

The enlargement of the spleen has been already mentioned as an almost constant symptom of the disease. Even during life some increase of the area of splenic dullness can usually be made out, and sometimes the spleen can be felt under the edge of the ribs.

The occurrence of the hemorrhages, which have already been referred to

under the pathological lesions, can often be demonstrated during life. The cutaneous ecchymoses can, of course, be seen, and the hemorrhages in the mucous membranes may give rise to hematemesis, bloody stools, bleeding from the female genitals, or epistaxis. The hemorrhages are due probably to the impaired nutrition and diminished resisting power of the vascular walls occasioned by the grave constitutional disturbance.

The condition of the urine in acute yellow atrophy is very interesting. The amount is either normal or slightly diminished, and the specific gravity is somewhat increased. Often there is a trace of albumen. We have already mentioned the presence of bile pigment. The point of chief interest, however, is one that Frerichs discovered and various others have since confirmed—namely, the great diminution of urea and the appearance in its place of other substances, which are likewise the products of the decomposition of albuminoid matter, and represent, in all probability, the first steps in the formation of urea. This explains, above all, the increased amount of ammonia in the urine, and the presence of leucin and tyrosin in the same. The characteristic crystals of the latter can sometimes be detected by the microscope in the urinary sediment (see Fig. 90). The crystals may also sometimes be obtained by allowing a drop of the fresh urine to evaporate slowly upon an object glass. The proper way, however, to demonstrate these crystals is to precipitate the urine with basic acetate of lead, to remove the lead from the filtrate by means of sulphureted hydrogen, and then to evaporate the filtrate thus obtained to the point of crystallization. There are some other abnormal constituents to be found in the urine besides leucin and tyrosin, but what their significance is we do not know. Among these are sarcolactic acid, oxymandelic (paraoxyphenylglycolic) acid, peptonoid substances, and large amounts of kreatin.

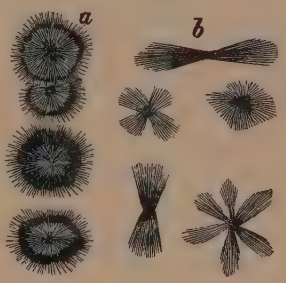


FIG. 90.—*a*. Leucin crystals. *b*. Tyrosin crystals.

It at once suggests itself that this disappearance of urea and appearance of ammonia, leucin, and tyrosin, which are regarded as preparatory stages in the formation of urea, gives valuable support to Meissner's and Von Schröder's idea that this substance is manufactured in the liver.

As to the other organs little need be said. Vomiting is very frequent in the second stage, as well as in the first. It usually ushers in the severe cerebral symptoms. The stools are, as a rule, clay-colored, as is usual in jaundice. There is generally constipation. The pulse is rapid, often reaching 140 to 160 beats per minute, and is also small and compressible. It is this acceleration of the pulse, contrasting with its usual slowness during the first stage, which, along with the vomiting, announces the onset of dangerous symptoms. The pulmonary signs are seldom marked, although there may be bronchitis or a pneumonia due to the inhalation of foreign substances. During the coma which precedes death respiration is usually hurried, and often deep and noisy. Sometimes it is irregular.

The temperature is at first approximately normal or slightly elevated. Toward the fatal termination there may be a subnormal temperature or the

temperature rises before death, and even sometimes grows higher after death, reaching 107.5° F. (42° C.) or more.

In case the disease attacks a pregnant woman, abortion or premature delivery is almost certain to occur.

The entire duration of the disease depends mainly upon the length of the first stage. This may be wholly absent, or may be brief, or may occupy several weeks. The duration of the second stage, reckoning from the occurrence of grave cerebral symptoms, is generally only a few days (two to four), rarely a week. The termination is almost invariably fatal. In a few cases recovery has been observed.

Diagnosis.—The diagnosis cannot be made till the second stage. The symptoms of the first stage are indistinguishable from those of simple catarrhal jaundice. With the development of the grave symptoms all doubt usually vanishes. The general course of the disease, the deep jaundice, the cerebral disturbances, the cutaneous ecchymoses, and the character of the urine, form a clinical picture resembling no other disease. The only cases where there can be any uncertainty about the diagnosis are the exceptional ones in which there is no jaundice. It is of practical importance to distinguish this disease from acute phosphorous poisoning (*q. v.*). The differential diagnosis is to be made from the history of the case and from the following factors:

ACUTE YELLOW ATROPHY OF THE LIVER	PHOSPHOROUS POISONING
1. Prodromes are usually present, but may be absent.	1. Prodromes are almost always present.
2. Jaundice develops early, sometimes becomes marked.	2. Jaundice does not appear until rather late, and it may be slight to the end.
3. Liver seldom enlarged, becomes atrophic from the start; pain in hepatic region not the rule.	3. Liver much enlarged until death, and very painful.
4. Maniacal delirium very frequent.	4. Seldom marked delirium.
5. Not infrequently fever, sometimes high fever.	5. Not infrequently fever is absent throughout.
6. Leucin and tyrosin often found in the urine.	6. Leucin and tyrosin found in urine exceptionally.
7. Spleen often enlarged.	7. Usually no enlargement of the spleen.

Treatment.—From what has been said, it may be gathered that the treatment of acute yellow atrophy of the liver is almost hopeless. Usually laxatives are employed, particularly calomel. The nervous symptoms are combated by an ice cap and baths and narcotics; the vomiting, by opium and bits of ice; and the cardiac weakness, by stimulants.

APPENDIX

PERNICIOUS JAUNDICE. CHOLÆMIA AND ACHOLIA

Reference has been repeatedly made in the preceding chapters to the possibility of the sudden supervention of grave nervous derangements in the course of various hepatic diseases. These nervous symptoms resemble one another so much in the different instances of their occurrence that we are forced to believe them always due to the same cause.

These symptoms are relatively most frequent when there is chronic biliary retention. Whether this retention be the result, for example, of obstruction of the common or the hepatic duct, or of stenosis of the biliary passages from a carcinoma involving the opening of the common duct into the duodenum or that duct itself, the patient may quite suddenly fall into a condition which in many respects corresponds to the second stage of acute yellow atrophy just described. Grave cerebral disturbances declare themselves, with delirium, convulsions, and coma. There are hemorrhages into the skin and into the mucous membranes, and in a few days the patient dies. Usually the end is attended with high fever. We have ourselves seen a temperature of 107.4° F. (41.9° C.) in a case of cancer at the duodenal extremity of the common duct. It is this group of symptoms which is usually termed pernicious jaundice; but almost precisely similar phenomena may suddenly appear in hepatic cirrhosis, when there is no great degree of jaundice, if any.

Exactly what produces these grave results in acute yellow atrophy and the other disorders just enumerated, we are not quite certain. Three theories have been propounded in explanation. The first theory, the latest champion of which is Leyden, attributes pernicious jaundice to cholæmia—that is, to the accumulation in the blood of the constituents of bile, and in particular of the biliary acids, as a result of absorption. This accumulation, it is said, is promoted by the paralyzing effect of the jaundice upon the activity of the kidneys. Opposed to this theory is the fact that these same symptoms may occur when there is no marked hepatogenous jaundice. Furthermore, it is doubtful if the constituents of the bile, especially the bile acids, can accumulate in such quantity in the blood that they can produce toxic effects.

Bouchard and other French physicians ascribe to the liver the function of rendering harmless the products of the decay of albuminous matters developing in the intestine, which may be absorbed and enter the circulation. Destruction of the hepatic cells would consequently lead to autointoxication with these intestinal ptomains. In Germany this doctrine has gained few adherents.

The view which we are most inclined to accept was first brought forward by Frerichs. He has given to the group of symptoms under discussion the name of acholia. These symptoms he ascribes to the pernicious influence of those substances which under normal conditions are manufactured by the liver into bile, but which in such cases accumulate in the blood and the tissues. As Frerichs himself has said, and we believe very justly, this view should be extended to include all other metabolic functions of the liver (urea production from ammonia and carbon dioxid, formation of carbohydrates from albumen, conversion of glycogen into sugar, etc.). There is no doubt that the processes of

metamorphosis are very active in the liver. That these should be incomplete when there is a severe anatomical lesion of the liver, or when there is persistent biliary obstruction, is very likely, and this limitation of activity might occasion an accumulation of all sorts of material in the blood. We may, therefore, regard acholia, using the word as Frerichs does, as entirely analogous to uræmia, which latter appears not only in diseases of the kidney but also when the ureters are obstructed. In this sense the name cholæmia would be preferable to the term acholia, to represent a condition analogous to uræmia—taking the word cholæmia to mean the intoxication of the blood with all sorts of substances due to the imperfect metamorphosis going on in the liver (hepatic autointoxication), and not, as Leyden formerly held, the saturation of the blood with the completely formed components of the bile.

The termination of cholæmia is almost always unfavorable. Only in exceptional cases have the symptoms disappeared after the occurrence of a profuse diuresis or marked catharsis. This suggests the indicated therapy—viz., catharsis, diuresis, hypodermoclysis, etc. At the post-mortem examination of cholæmic patients there is usually found marked fatty degeneration of the hepatic parenchyma as the anatomical expression of its suspended function.

CHAPTER VII

ICTERUS NEONATORUM

(Jaundice of the Newborn)

Frequently the normal red color of the skin in children changes on the second, third, or fourth day after birth to a distinctly yellow, jaundiced hue. The yellow tinge is deeper on the face and trunk than on the extremities. There are no special digestive or constitutional disturbances. Still, it may be taken for the rule that weaklings and premature infants more often present this jaundice than do vigorous babes. The abnormal hue is almost certain to vanish in a week or two, and leave no sequelæ. The termination is unfavorable in those instances alone where there is some special complication, not directly connected with the jaundice as such.

The ætiology is a disputed matter. A large number of theories of all sorts have been set up, no one of which to this day has gained universal acceptance. Formerly there was considerable tendency to regard the jaundice as hematogenous—that is, as due to the transformation of the pigment of broken-down blood corpuscles into bile pigment. Points which seemed to support this view are the light (not jaundiced) color of the urine and the yellow color of the stools (from bile). But more accurate examination has shown that the urine does contain biliary pigment, as do also the kidneys of such infants as happen to die during the existence of the jaundice; and the biliary acids have been clearly shown to be present in the serous transudations. It is therefore assumed at present by most authors that icterus neonatorum is hepatogenous; but how the biliary retention and consequent absorption are caused we do not know. Perhaps the bile is not ejected properly, from weakness, or the ducts may be narrow, or temporarily plugged by desquamated epithelium. Birch-Hirschfeld has

called attention to the fact that after birth there is a tendency to considerable passive congestion of the liver, with resultant œdema of Glisson's capsule and pressure upon the interlobular bile ducts. Probably more importance, however, attaches to a circumstance which Hofmeier has noted, that in the first days after birth many red corpuscles are disintegrated, and consequently there is a comparatively large secretion of bile. This might easily lead to congestion of the smallest bile ducts and so to icterus; but it should be said this view has its opponents. Quincke has an entirely different explanation of *icterus neonatorum*. He goes back to an old theory enunciated by Peter Frank. According to this, the jaundice of the newborn is due to the absorption by the intestine of a large amount of biliary coloring matter from the meconium in the large intestine. Now, if the *ductus venosus* remains open for some little time after birth, as it often does, this biliary pigment is carried not to the liver, but by means of the *vena cava inferior* directly into the systemic circulation. The meconium is rich in bile pigment, and moreover there is an abundant secretion of biliary coloring matter in the newborn, and in them bilirubin is not changed into urobilin in the intestine, and finally the renal secretion of the newborn is at first very scanty. Consequently, the absorption of the bile pigment from the intestine results in jaundice.

It is well to mention in conclusion that in very rare instances there is complete congenital stenosis, or even entire absence, of the larger bile ducts. Then deep jaundice comes on soon after birth, and is persistent. The children become extremely emaciated, and, after a few weeks, inevitably perish.

CHAPTER VIII

SYPHILIS OF THE LIVER

Ætiology and Pathology.—Syphilitic disease of the liver occurs both when the syphilis is acquired and when it is congenital. Congenital syphilitic disease of the liver may be diffuse or localized, and it causes a cellular infiltration in either case. If the changes are extensive, the organ is hard and considerably enlarged; or, if the newly formed connective tissue has undergone contraction, the liver is smaller than normal, and its surface is uneven. In some cases of hereditary syphilis, distinct gummata of considerable size have been observed.

In acquired syphilis, hepatic disease is one of the so-called tertiary symptoms, and does not usually develop, at least to any great extent, until several days after infection. Indeed, it may be a very late result. It may take the form either of a diffuse syphilitic hepatitis, or of circumscribed gummata or syphilomata. The diffuse hepatitis does not present essentially different anatomical appearances from those of ordinary cirrhosis, although in syphilis the changes are seldom so uniformly distributed throughout the organ. The gummata are the most characteristic, and the most important clinically. They may form separate tumors the size of an apple or even larger. The convex surface of the organ, particularly that portion near the suspensory ligament, seems to be a favorite location for the new growth. The same is true of the porta hepatis, where Glisson's capsule enters the liver. At the autopsy the

gummata have in most cases already begun to undergo contraction. If so, the liver is usually smaller than normal, and traversed in various directions by deep furrows, which divide it into lobes. These furrows are due to firm cicatricial bands, among the fibers of which we may sometimes find necrotic and cheesy vestiges of the gumma proper. Often there is evident syphilitic endarteritis in the smaller and sometimes also in the larger branches of the hepatic artery and portal vein.

Clinical History.—Circumscribed syphilitic changes in the liver often give rise to no symptoms whatever. It is only when the disease comes to disturb the portal circulation that a series of symptoms results, which, for evident reasons, may be analogous in all essential points to the effects of ordinary cirrhosis. Whenever the syphilitic growths contract so as to obliterate a large number of branches of the portal vein, or whenever a gumma happens to be so situated as to compress the trunk of the portal vein itself, then the well-known results of portal obstruction are inevitable, the chief being ascites and enlargement of the spleen. The disturbance of circulation often gives rise also to anorexia and digestive disorders. We once observed severe gastric and intestinal hemorrhages without gross changes in the corresponding mucous membranes—i. e., hemorrhage by diapedesis. Experience shows jaundice to be rare in hepatic syphilis, but it may appear when the lesions involve the larger bile ducts or a considerable number of the smaller biliary passages. It deserves mention that hepatic syphilis quite often causes severe pain, sometimes over the entire region of the liver and sometimes in just one spot. Pain is by no means felt in every case. With the pain there may be great tenderness on pressure.

On physical examination of the liver the results vary according to the form and the stage of the disorder. Sometimes the larger gummata may be plainly felt through the abdominal walls, usually as flattened hemispheres. Frequently, also, we can feel the edge of the enlarged organ, and can detect that the edge is less sharp than normal. In other instances the separate elevations and prominences can be made out. The area of dullness on percussion of course varies in different cases.

The course of the disease is usually tedious, and it may occupy many years. Probably, too, lesions exist in many cases long before there are any symptoms. Just as in cirrhosis, ascites is usually the first thing to attract the patient's attention. Improvement and temporary arrest of the disease are more frequent than in ordinary cirrhosis. Still, in most cases, if the lesions are at all extensive, the termination is unfavorable.

Of practical importance is a form of syphilis of the liver, which, however, has been little studied pathologically, and the course of which is more acute and is associated with a continuous remittent fever. Persistent febrile conditions of doubtful origin have been repeatedly observed in which an enlargement of the liver was demonstrable, and in which, after the administration of iodid of potassium, recovery ensued. These cases are explained on the basis of a gumma formation in the liver. We once observed a case of syphilis of the liver complicated by tuberculosis of the peritoneum—an interesting analogy to the combination of hepatic cirrhosis and peritoneal tuberculosis (*vide supra,* page 670).

Diagnosis.—The diagnosis is not always easy. Usually the objective changes in the liver, the ascites, and the enlarged spleen, indicate hepatic trouble, but we are often unable to determine just what the trouble is. Naturally,

the ætiological factors are of great importance. In a toper we would think rather of the common form of cirrhosis. If there is a syphilitic history, or if we find scars in the throat, irregularities in the surface of the bones, or other signs of a specific dyscrasia, we would naturally ascribe the hepatic disorder to the same cause. As to special signs, if the prominences on the liver are rather large in contrast to the smaller granulations of common cirrhosis, syphilis is somewhat more probable. Severe pain in the right hypochondrium also suggests syphilis rather than cirrhosis. It should also be considered that the course of hepatic syphilis is usually much more protracted than that of ordinary hepatic cirrhosis.

Treatment.—Specific treatment should first be tried. Mercury and potassium iodid should both be given, but probably the iodid is the more valuable of the two. These remedies can be successful only when the gummata are still in process of formation. Our therapeutic efforts produce no impression upon the cicatricial bands, the contraction of which is the main cause of derangement. Indeed, even the absorption of gummata may not always have a favorable influence upon the patient, because of the mechanical disturbance due to the consequent cicatricial change. Hence, in general, the results of antisyphilitic treatment are not very satisfactory. Only in the above-mentioned rare cases of tertiary syphilitic fever with gumma formation in the liver has the iodid of potassium usually any very good effect.

For symptomatic treatment, the reader is referred to the chapter on Cirrhosis of the Liver.

CHAPTER IX

CANCER OF THE LIVER AND BILE DUCTS

Ætiology and Pathology.—Primary cancer of the liver is very rare, but secondary or metastatic cancer of this organ is met with often. The chief explanation of this latter fact is the slowness of the blood current in the liver, which favors the deposition of the cancerous germs suspended in the blood.

Secondary hepatic cancer may be a sequel to primary cancer of any organ. It is most often seen, however, when the primary growth lies within the portal system, in the stomach, intestines, rectum, œsophagus, or pancreas. In some instances the projection of the primary growth into the lumen of a branch of the portal vein has been directly demonstrated, thus furnishing an obvious source for metastasis. The secondary cancers in the liver may be very numerous. They are found both within the organ and upon its surface. If superficial, they form flattened protuberances, which are often dimpled in the middle. If the new growth is extensive, the liver may be greatly enlarged, so as to occupy a great part of the abdominal cavity.

As we have said, primary cancer of the liver is very unusual. It may occur either in the form of separate large nodules, or as a more diffuse cancerous infiltration pervading the greater part of the organ. Histologically considered, the primary growths are of cylindrical-cell carcinoma, apparently originating from the epithelium of the minute bile ducts, but also, according to some authors, sometimes starting from the cells of the parenchyma.

Primary cancer of the larger bile ducts is of more frequent occurrence than genuine primary hepatic cancer, and therefore it is of more importance clinically. The gall bladder may also be the seat of primary carcinoma. From these sources may proceed abundant metastatic growths in the liver itself.

As to the ætiology of hepatic cancer we can be brief. The disease is most frequent in advanced life, from forty to sixty, following in this the general rule for cancer. Of special exciting causes, there is only one to be mentioned —that is, biliary calculi. As we have already remarked, the formation of cancer subsequent to cholelithiasis has been observed so many times that mere coincidence is out of the question.

Clinical History. Diagnosis.—Many small nodules of cancer, as well as large masses which are favorably situated, may exist in the liver without exciting any symptoms. If there is an undoubted primary cancer in another organ, such as the stomach, we must always remember the possibility of metastatic growths in the liver, but they cannot be proved to exist unless they alter appreciably the size or shape of the organ. Sometimes their existence may be inferred when we observe either ascites and enlargement of the spleen from pressure on the portal vein, or jaundice from pressure on the bile ducts.

On palpation we are often able to make out plainly one or more tumors in hepatic cancer. These tumors are in the region of the liver, and they are directly connected with it, as we can prove by marking out its limits by percussion and palpation. Another characteristic sign is that almost all hepatic tumors can be felt to move with respiration, on account of the inspiratory depression of the diaphragm pushing down the liver and all that is joined to it. Percussion over a hepatic tumor almost invariably gives flatness, in contrast to the muffled tympanitic resonance of many gastric tumors.

The most characteristic condition is not a very rare one; in it the liver is the seat of a very large number of cancerous nodules. In such cases the organ is usually much enlarged. Often we can detect by mere inspection a great, irregular prominence in the hepatic region, pressing forward the flabby, atrophied walls of the abdomen, reaching down to the level of the umbilicus, or even lower, and moving with respiration. On palpation we can feel most of the anterior surface of the liver, and the separate cancerous nodules scattered over it. These usually are as large as walnuts, or even apples, and they are often umbilicated. The lower or anterior margin of the liver can often be made out plainly, and it also is often the seat of nodules; and we can sometimes feel nodules on the lower surface of the organ.

The other clinical phenomenon in hepatic cancer have several causes: (1) The primary disease, such as cancer of the stomach; (2) the general cancerous cachexia, as shown by languor, emaciation, and possibly a slight œdema of the ankles; and (3) the possible compression of the blood vessels or bile ducts. Compression of the portal vein produces a moderate or even a large ascitic effusion. Even in these instances the spleen is seldom much enlarged as a result of the passive congestion, because the universal emaciation and anæmia counteract the tendency to increase in size. Jaundice is relatively more frequent in cancer of the liver than is ascites. It is caused by compression either of the hepatic duct or of the minuter bile ducts. On the other hand, however, we can easily see that hepatic cancer may exist without producing either icterus or ascites.

The differential diagnosis of hepatic cancer from cancer in other organs is sometimes extremely difficult. This is particularly true of pyloric cancer, and especially so when the pylorus has become adherent to the liver, which often is the case. Cancers of the omentum and of the colon sometimes simulate hepatic cancer, but they seldom move so decidedly in respiration as do hepatic tumors. Given a new growth in the liver, it is usually comparatively easy to distinguish between carcinoma and other tumors. In case of syphilitic new growths, we have to consider the previous history and other signs of syphilis, also the eventual contractions and diminution in size of the liver, the usually longer duration of the disease, and the general condition of the patient. Echinococci have, as a rule, a much more regular shape, like a flattened ball; they seldom occasion marked cachexia, and also have a much longer clinical history. Large abscesses are very rare in our climate, and they can usually be recognized by special objective conditions. Moreover, they cause fever (rigors), which is exceptional in carcinoma. Other primary tumors of the liver, such as sarcoma and adenoma, are very rare, and therefore of no great clinical importance. There is, however, one rare but important tumor of the liver which we must mention—viz., melanotic sarcoma, which may appear as a secondary growth in the liver. It may be of enormous extent, either diffuse or nodular, and secondary to melanotic sarcoma of the choroid or of the skin.

When we have decided that cancer of the liver is present, the question arises: Is it primary or secondary? In the first place, primary cancer here is so rare that the probabilities are in favor of a secondary growth. Not infrequently the primary tumor cannot be detected during life. Thus a small cancer of the stomach, or a flat œsophageal cancer, or carcinoma of the pancreas, are all easily overlooked. Cancer in the rectum may be detected by digital examination. If we find many nodules in the liver and no primary trouble elsewhere, then there comes the possibility that there is primary cancer in the gall bladder or the bile ducts. In rare instances palpation may discover the gall bladder in a state of cancerous degeneration close underneath the liver; but usually the viscus will be small and contracted, and the flat and ulcerating growth is not noticeable except from the inside. It is particularly in those cases of hepatic cancer in which there is at an early period great and persistent icterus, without evidence of carcinoma in any other organ, that we should be most apt to think of primary cancer of the bile ducts.

The duration of hepatic cancer is usually not prolonged. The first decided evidences of its existence no sooner present themselves than marasmus and cachexia rapidly develop. The fatal end comes in a few months, or at latest within a year.

The prognosis is hopeless. Treatment can avail only to alleviate somewhat the patient's sufferings.

CHAPTER X

ECHINOCOCCUS OF THE LIVER

Natural History and Pathology.—Inasmuch as it is the liver which suffers most frequently from invasions of the echinococcus, we will here state the main general points relative to the troubles produced by this parasite.

The *tænia echinococcus* (see Fig. 91) is a small tapeworm about 4 mm. long, and composed of four joints. It inhabits the intestinal canal of the dog. The head is small and bears a beak which is provided with some thirty to forty hooklets, arranged in two rows. Behind the circle of hooklets are four suckers. The last joint of the tænia is by far the largest, and contains some five hundred eggs. Man is infected by ingestion of the eggs. The great frequency of the echinococcus in Iceland is explained by the close intimacy of the Icelanders with their dogs. In Germany the echinococcus is most frequent in Mecklenburg and Pomerania.

FIG. 91.—(From HELLER.) Tænia echinococcus, enlarged. Above, at the right, echinococcus, of natural size.

If a human being has become infected, the embryo pierces the intestinal wall, and is carried with the blood current or the lymph current to some remote organ. Most of the eggs no doubt are destroyed in the stomach, as may be inferred from the fact that multiple echinococci are much less frequent than solitary individuals. In a great majority of cases the embryo passes through a branch of the portal vein into the liver and there fastens itself; but the echinococcus may be developed in other organs—including the lungs (*vide* Vol. I), the bones, the brain, the kidneys, and the heart. A hydatid cyst develops from the embryo, and is filled with a nonalbuminous fluid. The cyst is composed of an external cuticle of laminated structure, and an inner, parenchymatous layer, which contains muscular fibers and blood vessels. Surrounding the cyst, as it lies in the infested organ, there is gradually developed a thick capsule of connective tissue.

After the cyst has continued its growth for some four to six months, being now about the size of a walnut, there are generated upon the inner surface of the capsule, from the parenchymatous layer, so-called breeding capsules, containing numbers of echinococcus heads, or "*scolices.*" Each scolex has four suckers and a circlet of hooks. It can draw itself into the breeding capsule and also produce a prominence upon the outer surface of the latter (see Figs. 92, 93, and 94). The number of new-formed scolices in an echinococcus is very great. The sac grows very slowly up to about the size of a fist, or even larger; its wall has a pale yellow color, is transparent, and displays an extremely characteristic laminated structure. The firmly attached breeding capsules are visible from the outside through the walls as minute points.

FIG. 92.

FIG. 93.

FIGS. 92 and 93.—(From HELLER.) Echinococcus scolices, drawn in and turned outward.

The primary cyst often gives rise to secondary "daughter vesicles," and these to "granddaughter vesicles." Some of these are formed in the cuticle,

others from the breeding capsules. In man they generally grow inward—that is, are endogenous (*echinococcus hydatidosus*)—and finally become detached. Hundreds of them may sometimes be found free in the liquid contents of the cyst. In animals the daughter vesicles are more often exogenous (*echinococcus veterinorum seu granulosus*). A peculiar kind of echinococcus, which was formerly regarded as a kind of new growth, is that called by Virchow *echinococcus multilocularis*. This is a resistant tumor containing much connective tissue, and permeated with numerous small alveoli. The gelatinous contents of the alveoli consist of echinococcus vesicles. The growth probably originates from a single germ, which by persistent exogenous proliferation invades the lymph vessels (perhaps also the blood vessels and the bile ducts).

FIG. 94.—Echinococcus hooklets.

The growth of a hydatid cyst is very slow, and it may continue for years. At last, however, the echinococcus dies. The cyst then undergoes considerable though gradual contraction, and both walls and contents become fatty degenerated and calcified. In the mortar-like detritus it is usually possible to find some characteristic hooklets.

Clinical Symptoms.—As long as the cyst in the liver retains moderate dimensions, there is usually no discomfort. Frequently the cysts perish and become calcified without having ever attracted attention, and are found post-mortem merely by accident.

If the cyst becomes very large, it causes a sensation of pressure and pain in the hepatic region. In rare instances unusually large cysts, if situated on the convex surface of the liver, may crowd up the diaphragm so as to compress the lower portions of the lung and induce dyspnœa. In such cases we find on the anterior or posterior wall of the thorax an abnormal protuberance, with dullness and diminished respiratory murmur. If a large echinococcus grows downward into the abdominal cavity it occasions pressure, tension, and pain in the abdomen. Compression of the portal vein may lead to ascites, and compression of the bile duct, in rare cases, to jaundice. The tumor can usually be made out with ease by inspection and palpation. It is approximately hemispherical, smooth, not tender, and feels rather firm and elastic. There is often a sense of fluctuation, while the so-called hydatid thrill—to be elicited by quick pushes with the flat of the hand—is recognized distinctly in but few cases.

The condition may be complicated (1) by rupture of the echinococcus sac into neighboring parts, and (2) by secondary suppuration. Both occurrences may be apparently spontaneous, or the result of traumatic influences.

Of the possible directions in which rupture may take place, penetration of the pleural cavity should be particularly mentioned. It usually results in an acute empyema. Here we have to do partly with secondary infection occasioned by pyogenic bacteria, and partly, also, as it seems, with the influence of toxic substances (toxalbumens), which are present in the echinococcus fluid (*vide infra*). Penetration into the lungs with rupture of the echinococcus sac leads to severe pulmonary symptoms. The patient may cough up constituents of the bile and small echinococcus vesicles, or portions of the same. Perforation into the abdominal cavity occasions purulent peritonitis. If viable

daughter vesicles are set free in the abdominal cavity, they may settle on some fresh spot and continue to grow. As extreme exceptions are to be mentioned rupture of the echinococcus sac into the pericardium, the bile ducts, the intestine, the *vena cava,* and the pelvis of the kidney.

Suppuration of an echinococcus in the liver (developing occasionally after injuries, acute infectious diseases, etc.) leads to the clinical symptoms of hepatic abscess—increasing tenderness and fluctuation of the tumor, fever, aggravation of the constitutional symptoms, etc. Perforation of the abscess into neighboring structures may occur in the same way as above mentioned. Spontaneous recovery is possible, but infrequent. In general, suppuration of an echinococcus sac is a dangerous condition, unless there is surgical interference.

We must devote a few words to *echinococcus multilocularis* of the liver. As has been said, the tumor in this case is large, very firm and hard, and with a surface which is either smooth or rough. Tenderness on pressure is exceptional. On the other hand, there is a feeling of painful distention in the hepatic region. Comparatively often (H. Vierordt) there is jaundice, which may be extreme. In other cases the picture rather of a chronic portal obstruction develops (ascites, splenic enlargement, etc.). The bodily condition is fairly good for a long while, but later gradually increasing weakness develops. Secondary suppuration in the interior of the tumor also leads to fever and rapid loss of strength. The disease lasts two to three years, sometimes much longer.

Diagnosis.—The diagnosis of echinococcus of the liver depends chiefly upon the demonstration of a very slowly developing tumor in the liver, hemispherical, fluctuating, and painless, with no marked influence at first upon the general health. If the patient comes from a country where the echinococcus is particularly frequent, of course this renders the diagnosis easier. Formerly it was the custom to aspirate the tumor for diagnostic purposes. In this way we can, in many cases, obtain from the interior of the sac a light yellow nonalbuminous fluid, which often contains sugar and succinic acid, and in it sometimes, but of course not invariably, are found upon microscopic examination the characteristic hooklets, or little pieces of the laminated membrane. Lately, however, physicians have become much more cautious about puncturing echinococcus cysts, because the procedure is sometimes followed by symptoms which are probably due to the poisonous influence of the escaping contents of the cyst (*vide supra*). Not infrequently after puncture there occurs urticaria or severe toxic symptoms, such as collapse, vomiting, dyspnœa, diarrhœa, chills, and epileptiform attacks; and these may even prove quickly fatal, although occurrences of this sort are infrequent. They have brought great discredit upon the practice of aspiration for diagnostic purposes in case of echinococcus.

The interesting fact that an eosinophilia occurs in the blood of patients with echinococcus disease, as in infections with other animal parasites (tænia, etc.), is of considerable diagnostic significance.

The diagnosis of the multilocular echinococcus of the liver is always very difficult. The large hard tumor can be differentiated from carcinoma of the liver by the longer duration of the illness and the slighter constitutional involvement. On the other hand, the differential diagnosis from certain forms

of cirrhosis and syphilis of the liver is for a long time often impossible. Decisive for echinococcus in some cases is the perforation of the parasitic new growth through the diaphragm into the lung. This results in persistent chest symptoms—pain, cough, expectoration, and generally the discharge of a very characteristic peculiar, bile-stained, ochre-yellow sputum.

Treatment.—Inasmuch as internal remedies, such as iodin and mercury, are absolutely useless, the only treatment of echinococcus is by surgical means. In case the echinococcus tumor causes little if any discomfort, the patient is usually loath to consent to an operation, but it should be recommended in all cases, for it protects the patient from the possibility of later dangerous developments, and in itself is no grave matter. A description of the many methods of surgical treatment will be found in the text-books on surgery. In multilocular echinococcus cysts, surgical treatment is impossible. The therapy must therefore be purely symptomatic (puncture for the ascites, etc.).

CHAPTER XI

CIRCULATORY DISTURBANCES IN THE LIVER

1. **Hepatic Anæmia.**—Hepatic anæmia is seldom extreme except in cases of profound general anæmia, and it has no clinical importance, so far as we are aware.

2. **Passive Congestion.**—Passive congestion of the liver is of frequent occurrence and is of importance. It may arise subsequent to any disorder which disturbs the systemic circulation. It is oftenest seen in connection with heart disease, particularly mitral disease. It also follows pulmonary emphysema and chronic processes which result in contraction of the lungs. The liver is enlarged and engorged. The hepatic veins being situated in the center of the lobules, this central portion becomes darkly pigmented, while the periphery of the lobules seems lighter colored. The peripheral cells may even appear distinctly yellow, from a fatty infiltration which is not infrequent. In this way the cut surface comes to present that variegated appearance which has led to the name of "nutmeg liver." If the venous stasis be persistent, there is considerable atrophy of the hepatic parenchyma, involving especially the cells near the center of each lobule. Thus the liver is somewhat reduced in size in spite of the secondary increase of connective tissue, and its surface may become slightly granular. This is the "atrophic nutmeg liver" or "contracted liver due to passive congestion."

The clinical phenomena are chiefly those caused by the hepatic enlargement. If chronic cardiac disease, emphysema, or some analogous trouble has occasioned congestion of the liver, the area of hepatic dullness is increased, and frequently we can feel the edge or even a portion of the anterior surface. In well-developed cases the liver reaches a hand's-breadth or more below the ribs on the right side. If there is also tricuspid regurgitation (*q. v.*), the liver is usually much enlarged and can be felt to pulsate distinctly if the palm of the hand is laid upon it. Not infrequently in passive congestion of the liver there is jaundice, which may be slight or quite well marked. This is per-

haps due to the pressure exerted by the distended blood vessels upon the small bile ducts in the liver, and perhaps also to the secretion of a particularly viscid bile containing less water than normal, and tending to stagnate. As we have already mentioned, the peculiar mixture of jaundice and cyanosis in the complexion of many cardiac patients, is very characteristic. The secondary cirrhosis of the congested liver leads to ascites. We may suspect this condition of the liver in all cases of heart disease in which ascites is particularly well marked, in comparison with the slight œdema in other portions of the body.

Quite often the congestion, if great, produces subjective disturbances. There is a feeling of pressure and weight in the hepatic region, and if the capsule of the organ is tightly stretched there may be actual pain.

The prognosis and treatment depend, of course, upon the primary disorder.

3. **Active Hyperæmia.**—About active hyperæmia of the liver we have little definite information. Formerly there was a great deal said about it, as one of the conditions in "abdominal plethora." Active hyperæmia is most frequently assumed to exist in case of those who are good livers and of sedentary habit. In such, we are told, the temporary physiological hyperæmia which attends digestion passes on into a permanent congestion of the liver. Thereby the organ is enlarged, there are painful sensations in the right hypochondrium, digestive disturbances, and occasional slight jaundice. The abnormal condition just described is certainly often met with in practice, but it would seem hardly possible to draw a clear dividing line between active hyperæmia of the liver and other disturbances which give rise to similar symptoms. Such are chronic gastric and intestinal catarrhs, cardiac hypertrophy and functional cardiac derangement, with passive congestion of the liver, fatty liver, and incipient cirrhosis.

A prominent rôle in the production of active hyperæmia of the liver is also ascribed to the ingestion of such matters as are said to "irritate" the liver, like the various spices, coffee, and, above all, alcohol.

It should also be noted that the liver may be much engorged in many acute infectious diseases, particularly in pernicious malarial diseases and in typhus or typhoid fever.

It is also maintained that the hyperæmia may result from the cessation of hemorrhages elsewhere, such as the catamenia or bleeding from hemorrhoids. The facts that have been brought forward to sustain this view are none of them conclusive. We will mention that the "menstrual jaundice" which occasionally appears when the menses are scanty or absent has been referred to a vicarious hyperæmia of the liver.

It is, of course, impossible to make general statements about the course and duration of active hyperæmia of the liver. The treatment of the first variety mentioned—namely, that arising from an improper mode of life—demands careful regulation of the diet, exclusion of all alcoholic beverages, abundant exercise in the open air, such as horseback riding, and laxatives. We may order rhubarb, aloes, or a course of the waters at Carlsbad, Marienbad, Kissingen, or Homburg.

CHAPTER XII

ATROPHY, HYPERTROPHY, AND DEGENERATIONS OF THE LIVER

1. **Simple Atrophy of the Liver.**—Simple atrophy is not of rare occurrence, being seen in senile marasmus and in malnutrition from almost any cause. The degree of atrophy varies. The borders of the organ are much wrinkled. The lobules seem decidedly smaller than normal, and even the individual cells that still remain are atrophied and also usually deeply pigmented.

The condition does not of itself give rise to any special symptoms. The diminution in the perhaps demonstrable area of hepatic dullness is a sign too ambiguous ever to justify us in making from it a positive diagnosis of hepatic atrophy. Perhaps there is some value in the alleged lighter color of the stools, as indicating a diminished secretion of bile.

2. **Hypertrophy of the Liver.**—Even under normal circumstances the liver undergoes quite marked alterations in size. The exact point, therefore, where an abnormal hypertrophy begins cannot be set. Sometimes the autopsy reveals an unusually large liver, of which there had been no indications during life, and for which no cause can be made out.

There are certain diseases in which enlargement of the liver is found with comparative frequency: diabetes mellitus, chronic malarial poisoning, leukæmia, and sometimes rachitis. Topers quite often have enlarged livers, which, as a rule, present simple hypertrophic changes. Occasionally a liver has been reported as showing spots of localized hyperplasia, which may form flattened prominences upon the surface of the organ.

Hypertrophy is to be diagnosticated only when palpation and percussion give proof of an enlargement, and yet amyloid, hypertrophic cirrhosis, and other diseases which cause an increase in the size of the liver, can be excluded. The ætiology of the case should also be considered.

3. **Fatty Liver.**—This name is applied to excessive, diffuse, fatty infiltration of the hepatic cells. The size of the organ is increased. It is firm, anæmic, and of a uniform yellow color, both externally and upon section. The microscope shows that the cells of the parenchyma are filled with large and small globules of fat. The fat is most abundant toward the periphery of the lobules.

The causes of fatty liver are by no means clear. Sometimes it is found in cases of general obesity, where we may assume that the amount of fat which the liver receives as nourishment is abnormally great; but often we find a liver that contains comparatively little fat in those who have a well-developed *panniculus adiposus* and much fat in other organs. Topers may have a decidedly fatty liver. The occurrence of fatty liver in the cachectic, and particularly in the consumptive, is remarkable; and individuals suffering from cancer, marantic children, or patients with severe general anæmia, may also exhibit the same change. We have no intimate knowledge of the conditions that prevent, in such cases, the oxygenation of the fat which comes to the liver from the ingesta or from other organs.

We do not know that the fatty liver is in any way functionally impaired. The only clinical indication, therefore, of its existence is the increased bulk

of the organ. In phthisis we may sometimes feel pretty certain that the liver is fatty if an increase in bulk can be demonstrated, and if other causes for this enlargement, such as amyloid degeneration, appear improbable. If the anterior edge of a fatty liver can be felt, it is usually found to be noticeably thick and blunt.

The treatment of fatty liver is to combat the original disease.

4. **Amyloid Liver** (*Waxy Liver*).—Amyloid degeneration of the liver is almost invariably a part of extensive amyloid disease, involving also the spleen, kidneys, intestine, and other organs. The disease occurs chiefly in certain cachectic conditions, such as chronic suppuration, as in caries and persistent empyema, and also in chronic pulmonary tuberculosis and constitutional syphilis.

The amyloid liver is usually increased in bulk. The organ may even become almost double its normal size. It feels very firm and hard, its surface is perfectly smooth, and its edge is slightly thickened. The cut surface presents a characteristic grayish-brown "waxy" appearance.

The microscope shows that the degenerative process attacks chiefly the walls of the hepatic capillaries, the hepatic cells proper showing infrequent and slight amyloid changes. Very often the cells of the parenchyma are atrophied and somewhat infiltrated with fat.

The diagnosis of amyloid liver requires (1) the demonstration by palpation and percussion of hepatic enlargement. We can often feel a large part of the anterior surface and the margin of the hard and firm organ. The liver may reach as low as the level of the umbilicus. The diagnosis further demands (2) that some disease which predisposes to amyloid be present, and (3) that there be evidence of the degenerative process in other organs: the spleen should be enlarged, and the kidneys secrete an abundance of albuminous urine.

The other symptoms, as well as the prognosis and treatment, are determined mainly by the nature of the causative affection. The use of iodid of iron, iodid of potassium, the alkaline carbonates, and ammonic chlorid, respectively, has been recommended as particularly efficient, but therapeutic claims of this sort do not withstand a rigorous criticism. The chief point is the general improvement of nutrition by careful diet and nursing. Further particulars about amyloid disease in general will be found in the chapter on Amyloid Degeneration of the Kidney.

CHAPTER XIII

ANOMALIES IN THE SHAPE AND POSITION OF THE LIVER

1. **Corset Liver.**—The constant pressure of the lower ribs against the liver, as a result of tight lacing, often produces an atrophy of the hepatic parenchyma from pressure, as shown by a deep furrow crossing transversely the anterior surface of the organ. This "corset furrow" lies chiefly in the right lobe. Its usual situation corresponds to the margin of the ribs, and the atrophy may be so extreme that the liver is divided into a large upper part and a small, usually roundish, lower portion, connected by a narrow isthmus of tissue. At the

atrophic place the connective-tissue capsule of the liver is almost always much thickened. Often the lower section can be bent upward as if attached by a hinge.

This deformity of the liver is found quite often in elderly females, and rarely in men, as in soldiers. Unless extreme, it cannot be detected during life, and it causes no discomfort. Even the bad cases do not, as a rule, occasion any special symptoms; but they can be clearly made out if the abdominal walls are lax. The deep transverse furrow can be felt, and also the lower section, with its usually blunt edge. Particularly in the case of old women we must bear this condition in mind, else we might easily confound it with some enlargement of the liver, such as amyloid or passive congestion, or even new growths.

In some few cases a well-marked corset liver seems to cause especial symptoms. It occasions a constant feeling of pressure and tugging in the region of the liver. Sometimes there are attacks of violent pain, with peritoneal irritation and vomiting, and a mild degree of collapse. These have been regarded as the result of temporary congestion and swelling in the portion of the liver below the constriction, but we should view this explanation with reserve, and should bear in mind that in corset liver gallstones are remarkably frequent (*vide supra,* page 670), and that consequently the symptoms mentioned may also be connected with cholelithiasis. The treatment of such attacks, even when the diagnosis is doubtful, will in every case consist chiefly in absolute rest in bed, the employment of poultices or perhaps of an ice bag, and a restricted diet. If there is violent pain, we must resort to narcotics.

2. **Movable Liver.**—This name has been applied to a condition exceptionally seen in women with very lax abdominal walls, in which the liver sinks deep down into the lower portions of the abdominal cavity, probably as a result of an unnatural length of the suspensory ligament. The organ can be distinctly felt in its new situation, and can usually be brought back to a normal position with tolerable ease by means of external pressure. It is invariably very movable, and it can be seen to change its place when the patient lies upon the side. In most cases there are at the same time the indications of universal enteroptosis, with which we have already become acquainted (see page 565). The interpretation of the symptoms present, such as pain and digestive disturbance, is consequently difficult in most cases, especially if the patient is also neurasthenic and hysterical, which is not infrequently the case. The reader may compare on this point the chapter on movable kidney. Treatment is governed by the same principles as in the other forms of enteroptosis. Above all, trial should be made of a suitably applied and well-fitting bandage.

CHAPTER XIV

SUPPURATIVE PYLEPHLEBITIS

(Purulent Inflammation of the Portal Vein and its Branches)

Ætiology.—Purulent pylephlebitis is seldom a primary, idiopathic disease. In most instances it is due to the propagation of a suppurative inflamma-

tion of neighboring tissues to the walls of the vein. The main trunk of the portal vein is rarely directly attacked. Usually the process originates in the hepatic branches of the vein or in the veins of the portal system, and thence extends to the larger vessel.

Perityphlitic abscess is the most frequent source of suppurative pylephlebitis. The inflammation involves a mesenteric vein, and thence extends upward. Other causes are gastric ulcer, intestinal ulcers, as in dysentery, splenic abscess, and purulent inflammation at the porta hepatis or within the liver itself, as in abscess due to gallstones. The mode of production in these cases is precisely analogous to that in perityphlitic abscess, but they are rare.

A special form of pylephlebitis is observed in the newborn. Here the inflammation originates in the umbilical vein, and we need hardly say that the cause is a suppurative infection through the navel.

In rare instances it has been found that pylephlebitis has resulted from the penetration into a vein of some foreign body that had been swallowed, such as a pin. Here, too, the true factors in producing the inflammation are, of course, the bacteria which adhere to the foreign body.

Pathology.—Where the inflammation has attacked the vascular walls, the vein is thickened, and often the surrounding connective tissue is infiltrated with pus cells and mottled with minute ecchymoses. If the vein is cut open, the intima is seen to be opaque and often superficially ulcerated. The lumen of the vessel is filled with a thrombus, which is usually to a great extent in a state of purulent softening, so that offensive purulent fluid flows out. The course of events is as follows: First, the wall of the vein becomes inflamed. As a consequence of this, a thrombus forms at the same place. The bacteria penetrate this thrombus and occasion its purulent softening.

The extent of a pylephlebitis naturally varies in different cases. As a rule, little fragments become detached from the thrombus and enter the liver, producing metastatic abscesses. Secondary suppuration may occur also in the lungs, kidneys, brain, and joints, so that we have all the anatomical characteristics of a general pyæmia.

Clinical History.—Inasmuch as the primary, causative disease may be very different in different cases, it is impossible to delineate the disease comprehensively. It is, however, frequently ushered in by a number of symptoms, which render a diagnosis possible, at least in some cases, if the original disease has been recognized.

The symptoms of suppurative pylephlebitis are in part due directly to the local disease itself, and in part are occasioned by the general pyæmia. One of the local symptoms is pain in the epigastrium. This is rare. It may radiate downward or laterally, according to the starting place and extent of the inflammation. An inevitable result of the portal thrombosis is portal obstruction. The spleen becomes considerably swollen, and, if the disease be not too quickly fatal, there is an evident effusion into the peritoneal cavity. The splenic enlargement cannot be regarded as due merely to venous stasis, but is in part the "acute splenic tumor" of constitutional septic conditions. If the inflammation spreads from the branches of the portal vein to the neighboring bile ducts, jaundice results. Sometimes it is due also to the hepatic abscesses, or to a gallstone which happens to cause trouble simultaneously. Now and then there is no jaundice whatever.

Of the pyæmic symptoms, hepatic abscesses come first. They are due, as we have said, to the conveyance of infectious matter directly into the liver by emboli. The one almost constant sign of their occurrence is a decided enlargement of the liver. When there are no hepatic abscesses, the organ usually retains its normal bulk.

The course of the fever is very characteristic. As in other pyæmic conditions, there are almost invariably great elevations, to 106° F. (41° C.), or higher, accompanied by rigors, and followed by marked remissions, with profuse perspiration. These onsets of fever occur at irregular intervals, either daily, or every two or three days.

There are at the same time indications of constitutional septic infection, which keep increasing in severity. The pulse grows rapid and small. Intelligence is impaired. Somnolence and delirium come on, and the strength rapidly fails.

There are other symptoms. Vomiting is frequently seen. The bowels are seldom constipated, but usually relaxed. The dejections may contain blood, because of the venous stasis. In some cases the inflammation extends so as to produce a fatal general peritonitis. It is noticeable that the urine is generally scanty, and the amount of urea is strikingly diminished.

The disease usually runs a rather acute course. On the average, it lasts about two weeks, but it may occupy three or four weeks, or even a longer period. It is invariably fatal. At least, no cases of recovery are known.

The diagnosis can sometimes be made with considerable positiveness. In other instances it is impossible to exclude other pyæmic conditions, or abscess due to gallstones, etc. Important factors are the origin of the trouble—if it can be made out—the pyæmic rigors, the enlargement of the spleen and liver, jaundice, epigastric pain, and the evidences of general sepsis.

Treatment is unfortunately almost entirely useless. The fever is not affected even by large doses of quinin. All we can aim at is to support and relieve the sufferer as far as possible.

CHAPTER XV

THROMBOSIS OF THE PORTAL VEIN

(Chronic Adhesive Pylephlebitis. Pylethrombosis)

Ætiology and Pathology.—Like suppurative pylephlebitis, chronic portal thrombosis is not an independent disease, but is the sequel of a great variety of other pathological conditions. Marantic thrombosis is of rare occurrence, and is usually formed toward the close of life, so as not to be of practical interest. Apart from this, almost all cases of thrombosis of the portal vein are due to a compression and constriction of the trunk of that vessel or one of its main branches. This most often occurs in certain chronic hepatic diseases which involve a mechanical stenosis, either of the smaller branches of the portal vein within the liver or of the vein itself, with resulting coagulation of the blood within it. Chief among these diseases are cirrhosis and syphilis of the liver, which have repeatedly been observed to entail portal thrombosis; but other

diseases in the neighborhood of the vein may produce a similar effect. Portal thrombosis may also arise through compression of the vessel by a chronic inflammatory hyperplasia of the connective tissue at the porta hepatis. This is illustrated in chronic peritonitis, whether circumscribed or diffuse, an example of the former being sometimes seen as an effect of duodenal ulcer.

It was formerly held that many forms of so-called "lobulated liver" were due to a primary adhesive pylephlebitis. This is erroneous. These cases are probably all due to some primary hepatic disease, usually syphilitic. The size of the liver is little influenced by obstruction of the portal vein, even if long continued, for the hepatic artery suffices to supply all the blood required by the organ.

The anatomical changes in pylethrombosis do not differ essentially from those seen in thrombosis of any other vein. If fresh, the thrombus is still red; later it grows harder, paler, and more friable. If the thrombosis has existed a long while, the clot becomes completely organized. We have observed this even in the main trunk of the portal vein.

Clinical History.—The symptoms of portal thrombosis are those occasioned by the obstruction, and therefore such as we have already repeatedly mentioned, in connection with various hepatic diseases. The intensity and extent of these results, as well as the time occupied in their development, depend, of course, upon the place and size of the clot. If it is the portal vein itself which is attacked, and if the thrombus is extensive enough to obstruct the flow of blood, then the signs of venous stasis are evident throughout the portal system. The spleen becomes much enlarged, as can be easily demonstrated by percussion and palpation. Soon ascites appears as a result of the passive congestion of the peritoneal veins; and from a similar condition of the gastrointestinal veins arise catarrhal disorders, such as diarrhea, or, not so very exceptionally, there is repeated gastric and intestinal hemorrhage.

As we have seen, a collateral circulation may be developed (*vide* page 689), by which the venous blood of the portal system is enabled to reach the systemic veins. This explains why some of the symptoms of venous stasis may temporarily (perhaps permanently) vanish. We saw one case of portal thrombosis, the sequel to what was apparently a syphilitic disease of the liver, in which quite a large ascitic effusion appeared some six or seven times at intervals of three to six months, and under proper nursing and treatment, without aspiration, as often disappeared. The patient did not die till the illness had continued six years, and tapping had been required in all some fifteen times. At the autopsy the trunk of the portal vein was found to be converted into a fibrous cord with a lumen which barely admitted a knitting needle.

In simple pylethrombosis there are no local symptoms such as pain. The condition of the liver depends upon the primary disease. It is possible that a moderate atrophy of the entire organ might at length ensue if the portal blood were permanently cut off from it. But, as we have said, any cirrhotic changes, or any "lobulation," are not to be regarded as the result, but as the cause of the thrombosis, or at least as related to the cause.

The course and duration of the disease are according to the nature of the original, causative trouble. No general statements can be made.

The diagnosis of thrombosis of the portal vein is usually extremely difficult, and it can really hardly ever be made with absolute certainty. We may, in-

deed, recognize readily that there is some decided obstruction to the portal circulation; but whether this be due to a thrombus, or to compression of the portal vein, or to the obliteration of a large number of the smaller branches of that vein within the liver, we cannot always determine. Pylethrombosis may be regarded as probable, if no other cause of the portal obstruction seems likely, and if certain circumstances, such as a previous syphilis, point to the possibility of a portal thrombosis.

The prognosis is always unfavorable, although there may be, as we have said, great temporary improvement. Treatment must be symptomatic, and it follows in the main the principles set forth under cirrhosis of the liver.

APPENDIX

DISEASES OF THE PANCREAS

Inasmuch as the pancreas is almost entirely inaccessible from the standpoint of direct clinical examination, attempts have been frequently made of late to discover methods and signs that point, at least in general, to a disturbed function of that organ (functional pancreatic diagnosis). On account of the great difficulty of this problem, the results are mostly very indefinite. Still, the following practical and important facts may be considered at the present time:

1. Boldireff has found that upon the introduction of large amounts of fat into the stomach, a regurgitation of bile, intestinal juice, and pancreatic secretion into the stomach occurs. Volhard has attempted to utilize this fact in the diagnosis of pancreatic disease. Through a stomach tube he introduces into the empty stomach of the patient 7 ounces (gm. 200) of oil, and after half an hour siphons off the gastric contents and examines for trypsin. This has been demonstrated repeatedly and proves, at least if positive, that the pancreatic function is maintained.

2. The attempt has been frequently made to draw conclusions as to the digestive function of the pancreas from the condition of the stools. The conditions are quite complicated on account of the mixture of the various digestive juices (bile, intestinal juice). Still, we may say that very fatty stools—so called steatorrhea—particularly if icterus be absent—are always suggestive of pancreatic disease. It is also worthy of note that in the absence of the pancreatic function a diminished fat-splitting occurs—i. e., a diminution of the fatty acids and soaps present in fatty stools to below seventy per cent of the total amount of the fat present.

3. Worth noting is the not infrequent occurrence of glycosuria, or, at least, of "alimentary glycosuria," in pancreatic disease—i. e., the advent of glycosuria after the administration of 3.5 ounces (gm. 100) of dissolved dextrose.

4. The attempts made by E. Müller and Schlecht in my Breslau Clinic to demonstrate the presence of pancreas trypsin in the stools themselves are most definitive. After the rectum has been cleansed by an enema, the patient receives a test meal of 5 ounces (gm. 150) of meat and 5 ounces (gm. 150) of mashed potatoes, and about half an hour later a cathartic, as, for instance, calomel and

purgen, āā 3 gr. (gm. 0.2). The subsequent stools coming from the small intestine are now examined for their trypsin content, with the aid either of the digestion test on the so-called Löffler plate, or, what is simpler in practice, of Rumpel's gelodurat capsules filled with powdered wood charcoal. Such a capsule is placed in a small glass with about 0.5 ounce (10 to 15 c.c.) of triturated feces, and allowed to stand at a temperature of 98.6° F. (37° C.). If the stool contains trypsin, the capsule is dissolved, and the charcoal which is discharged colors the entire contents of the glass black. If trypsin is absent, the capsule will not be dissolved.

5. An interesting and peculiar urine reaction is the so-called Cammidge reaction. Its significance is still not entirely clear, but a positive reaction is supposed to speak for a disease of the pancreas. We cannot enter here into the details of this rather complicated reaction (boiling of the urine with HCl, neutralizing with carbonate of lead, addition of phenylhydrazin, acetate of sodium, and acetic acid, and a precipitation after twenty-four hours of delicate crystalline needles, which Cammidge takes to be osazone). The test is accepted as a proof of the presence of decomposition products of fat (glycerin) in the urine. In general, control tests of this reaction which have been made up to date have given satisfactory results, so that it seems that the reaction is not without practical significance.

The most important anatomical lesions of the pancreas which are known up to the present time are as follows:

1. **Atrophy of the Pancreas.**—The organ may share in a general marasmus. There is sometimes also extreme atrophy of the pancreas in those who have died of diabetes mellitus (*q. v.*).

2. **Hemorrhages Into the Pancreas.**—Small hemorrhages occur in the pancreas in cases of general hemorrhagic diathesis, marked passive congestion, severe acute constitutional infection, and trauma of the abdomen. These do not, as a rule, cause marked clinical symptoms. On the other hand, Klebs, Zenker, and others describe cases in which a more or less extensive pancreatic hemorrhage has been found at autopsy as the only demonstrable cause of death. The patients were previously in apparently perfect health and were vigorous, although usually corpulent individuals, and they died suddenly. Perhaps it was the influence of the hemorrhage upon the semilunar ganglia or the solar plexus which occasioned the speedy death. At any rate, we find in such cases the cavities of the heart of usual size and empty, while the abdominal vessels are distended with blood. The special causes which lead to the hemorrhage are not always clear. In general, we probably must surmise a primary disease of the blood vessels (syphilis, atheroma). It is noteworthy that a comparatively large number of cases have occurred in hard drinkers. Many cases also seem to be similar to those first described by Balser. These have an uncertain ætiology, perhaps infectious, and exhibit a multiple necrosis of fat and of the pancreatic tissue—there being numerous minute necrotic foci in the pancreas and in the fat tissue of the mesentery. Extensive disorder of this kind is observed particularly in obese persons, but is not confined to them. Sometimes in cases of pancreatic hemorrhage death is extremely sudden and apoplectiform. In other cases the symptoms last from twelve to twenty-four hours before the fatal termination. The patient suffers from violent abdominal pain, vomiting, abdominal distention, and general collapse; treatment is purely symptomatic.

3. **Pancreatitis.**—In rare cases primary acute pancreatitis, usually hemorrhagic, has been observed. It begins with violent colicky pains in the epigastrium. Vomiting and collapse soon follow. The pulse grows small, the extremities become cool, and death is speedy. At the autopsy the pancreas is found to be much enlarged, and mottled with ecchymoses, or it even presents scattered foci of suppuration. The ætiology is unknown. It is most likely that the germs which excite the inflammation originate in the intestines. There should be a sharp distinction made between primary pancreatic hemorrhage and acute hemorrhagic pancreatitis, but formerly these two conditions were often confounded. Secondary abscesses of the pancreas are not infrequent in pyæmia.

[Fitz, in the Middleton-Goldsmith lecture for 1889, threw much light on the ætiology and diagnosis of acute pancreatitis, which he subdivides into three anatomical forms—the hemorrhagic, the suppurative, and the gangrenous. He shows that the affection is not so rare as has been supposed; that its victims are usually in middle life, fat, and good livers; that it commonly originates by the extension of a gastroduodenal inflammation along the pancreatic duct.

"If the case does not end fatally in the course of a few days, recovery is possible, or a recurrence of the symptoms in a milder form takes place, and the characteristics of a subacute peritonitis are developed."

In the differential diagnosis irritant poisoning, perforation of the digestive or biliary tracts, and acute intestinal obstruction are to be considered. The location of peritonitic symptoms, their suddenness of onset, the absence of apparently sufficient cause, and the age and habit of the individual are important points.]

Chronic interstitial pancreatitis sometimes results from the extension of chronic inflammatory processes affecting neighboring parts. Friedreich states that it sometimes is a primary disease in topers. Syphilitic lesions of the pancreas have been observed, occasioning contraction and induration.

There are no characteristic clinical symptoms which correspond to these various changes. The symptoms observed in the cases belonging in this category are digestive disturbances, indefinite pain, bodily weakness, emaciation, and anæmia, and they are so ambiguous that a diagnosis of the true condition is scarcely ever possible.

We find a peculiar atrophy and induration of the pancreas associated with hemosiderosis (pigmentation by a ferruginous pigment) in the so-called bronze diabetes (*diabète bronzé*). This deserves brief mention here. It is a remarkable combination of diabetes (marked glycosuria) with dark pigmentation of the skin, dependent upon the deposit of this same ferruginous pigment in the skin. Usually, there is present at the same time a cirrhotic induration and enlargement of the liver, which is also associated with hemosiderosis, and not infrequently, too, with the signs of portal obstruction (enlarged spleen, ascites). The causes of this remarkable and rare disease are still entirely unknown.

4. **Cysts of the Pancreas.**—After closure of Wirsung's duct by scars, concretions, or the like, cysts of the pancreas may form, as a result of the damming up of the secretion. These may become so large as to be felt as great tumors through the abdominal walls. In other cases we seem to be dealing with cystic neoplasms. The cysts, as a rule, protrude between the stomach and transverse colon, although the greatest variety of other relations between the cysts and these organs may occur. A cure is possible only by operation. Rarely, spaces are found in the neighborhood of the pancreas filled with a thick, slimy fluid.

These seem to be associated with the above-mentioned peculiar multiple necrosis of fat and pancreatic tissue, but with regard to them further observations are desirable.

5. **Cancer of the Pancreas.**—Primary cancer is the most frequent, and therefore clinically the most important disease of this organ. It is usually of the medullary variety, though occasionally colloid. As a rule, the new growth is situated in the head of the pancreas. It may involve neighboring parts by direct extension, and a great many organs by metastasis; for example, the liver, peritoneum, and lymph-glands.

The clinical symptoms of cancer of the pancreas are very seldom so decided as to justify a positive diagnosis. Sometimes the secondary nodules can be detected in the liver, peritoneum, and elsewhere. Then we are left in doubt about the seat of the primary growth. Or the primary tumor may be plainly felt through the abdominal walls; but then we can hardly ever exclude cancer of the stomach or of the omentum and neighboring parts.

The symptoms of pancreatic cancer, as a whole, resemble closely those occasioned by most cancers of abdominal organs. Usually the patient is elderly. The first symptoms are weakness, emaciation, and indigestion, or they are the result of compression. Often there is complaint of a persistent dull pain in the epigastrium. If the portal vein is pressed upon by the tumor, ascites appears. If the common duct is compressed, there is jaundice. Icterus is an especially frequent symptom of pancreatic carcinoma.

The *diagnosis* is difficult. It can be made with a certain degree of probability when there is a slowly growing tumor in the region of the pancreas, without enlargement of the liver, but accompanied by jaundice, deeply seated epigastric pain, glycosuria, and the above-mentioned more or less distinctly marked pancreatic symptoms—viz., fatty stools, imperfect digestion of albumen and starch and glycosuria. Usually, however, the clinical picture is by no means so characteristic, and it is often impossible to avoid confusing cancer of the pancreas with cancer of the stomach, duodenum, gall bladder, or other organs.

The *prognosis* is absolutely bad. With increasing marasmus, the patient usually dies at the end of six months or a year.

The *treatment* is merely symptomatic, with the aim of lessening the patient's suffering.

6. **Pancreatic Calculi.**—In the excretory ducts of the pancreas are sometimes found calculi of small or moderate size, consisting essentially of carbonate and phosphate of lime. The cause of their formation is as little settled as that of gallstones (which see). Because of the obstruction caused by these calculi we have secondary dilatation of the ducts, secondary contraction and atrophy, and in some cases secondary inflammation and suppuration; these probably result from processes similar to those with which we have become acquainted while considering biliary calculi. There may be no symptoms. Sometimes, however, there is violent pain, either continuous or paroxysmal, also digestive disturbances, and occasionally the above-mentioned changes in the dejecta, and glycosuria. In only a very few cases has it been possible to find pancreatic calculi in the feces. An absolute diagnosis can rarely be made. As a therapeutic measure Eichhorst has recommended the stimulation of the pancreatic secretion by means of injections of pilocarpin; as a rule, treatment will be purely symptomatic.

V. DISEASES OF THE URINARY ORGANS

SECTION I

Diseases of the Kidneys

CHAPTER I

GENERAL PRELIMINARY REMARKS UPON THE PATHOLOGY OF RENAL DISEASE

Although some knowledge of the occurrence and significance of renal affections had been acquired even by the older physicians, still, the service of having pointed out the frequency of these diseases, and of having clearly recognized their most important anatomical forms and their chief clinical symptoms, belongs undoubtedly to the English physician Richard Bright, who was born in 1788 and died in 1858, as physician in ordinary to Queen Victoria. Bright's first work on this subject appeared in the year 1827. In this he brought forward the special discovery that, in many cases of general dropsy, which are associated with the secretion of an albuminous urine, a primary affection of the kidneys must be regarded as the true cause of the disease. Since then, the disease described by him has been almost universally called "Bright's disease" ("*Morbus Brightii*"), a name still much employed, but in whose stead the anatomical terms would be more proper, since many forms were previously classed under it which, according to our more accurate present knowledge, must be separated.

Bright's statements were either confirmed or expanded in subsequent times by many other observers. Christison, Osborne, and R. Willis in England, and Rayer and M. Solon in France, were the chief students in renal diseases. Frerichs published the first great work in Germany in the year 1851. His division of Bright's disease into three different "stages," based on Reinhardt's histological investigations, was for a long time quite generally accepted, until gradually further clinical experience showed that it was untenable. A more accurate division of renal diseases was substituted for it first in England (Johnson, S. Wilks, and others), and then in Germany (Traube, Bartels). However admirable these labors, especially the work of Bartels in 1871, renal pathology fell into subjection to theories, with which the facts of experience could be harmonized only by force. Only of late years has a natural theory of renal diseases, derived from general pathological observations, become accepted—a theory which is based chiefly upon the anatomical work of Weigert. There has

also been special research work done in the more accurate determination of the disturbed function of the diseased organ (v. Koranyi, etc.).

The following presentation of renal pathology is intended to show that, from a clinical standpoint as well, the renal diseases may be viewed comprehensively as a unit, and this not in a schematic way, but with due regard to actual conditions:

The chief reason why the kidneys are so often diseased, either alone or in conjunction with other organs, is to be found in the fact that the body must eliminate all forms of injurious matter, which circulate in the blood, in great part by the kidneys. Consequently the action of any injurious substance is often manifested chiefly in the kidneys, and they must, in a certain measure, suffer for the service which they do the rest of the body. According to their nature and character, the injurious substances, which are here to be considered, are divided chiefly into two great groups—the chemico-toxic and the organized infectious substances. At the same time, it should be noted that in case of infection it is comparatively exceptional for the germs themselves to reach the kidneys and there fix themselves. Most cases are referable to toxins, which are formed in the body as the result of the infectious process, and, being excreted through the kidneys, produce a nephritis. In this way the kidneys may be involved sympathetically after the ingestion of many poisons, both organic and inorganic, and also in the great majority of all the infectious diseases. In these cases, of course, as we shall see later, certain chemical and infectious poisons exert their action in a particularly frequent and in a particularly severe or definitely characterized fashion. Besides these forms of origin for many renal diseases ("hematogenous nephritis"), which are the chief ones to be considered, we must consider other causes of disease which are much rarer. One way in which the morbific agents may also enter is especially important—namely, from the lower urinary passages, the bladder, and pelvis of the kidney, upward into the kidney. In this way those renal diseases arise which come on secondarily to cystitis, pyelitis, etc. Finally, of course, disturbances of circulation and mechanical injuries may also make themselves manifest in the kidneys.

The clinical symptoms which are caused by the different forms of renal disease are referable only in very small part directly to the diseased organ itself. In renal diseases characteristic subjective local symptoms—such as local pain—are rare, and the anatomical position and the physiological conditions of the kidneys make it almost impossible to discover any changes in their size, their physical consistence, etc., by a direct objective examination. In the diagnosis of renal diseases we are therefore confined chiefly to the investigation of two group of sympoms: in the first place to the examination of the secretion from the kidneys, the urine, whose character, as we know by experience, may be materially altered when there is renal disease; and, in the second place, to the discovery of certain phenomena in other portions of the body, which are immediately dependent upon the renal affection. Since both the pathological changes in the urine, and the symptoms in other organs occurring in renal affections, have much in common in almost all the forms of renal disease, it is advisable first to describe the main features, at least, of the general symptomatology of renal disease. We shall then be obliged, in the following chapters, to mention only the precise circumstances of the occurrence and onset of each symptom—the general significance of the symptoms being already known.

1. ALBUMINURIA

The most constant symptom, which in many cases, even by itself, renders the diagnosis of a renal affection possible with complete certainty, is albuminuria—that is, the appearance of albumen, and especially of serum albumen and, in much smaller quantity, serum globulin, in the urine. Strictly speaking, every albuminuria must be regarded as somewhat pathological. From recent investigations (Leube, Fürbringer, Pavy, Heubner, and others) we know that in some cases the urine may, for a short time, contain a very slight amount of albumen even in healthy persons, especially after physical exertion, emotional disturbance, a cold bath, a hearty meal, etc.

As we may assume from the exciting causes, there does exist a slight and rapidly disappearing disturbance of the kidney, which produces no further sequelæ. In such cases we may speak of a temporary and insignificant, but not really of a "physiological" albuminuria. We must also mention here the slight albuminuria which is not infrequently found in newborn infants, and which disappears after eight to ten days.

Intermittent, Orthostatic Albuminuria.—The conditions are similar in the rather frequent cases of so-called intermittent albuminuria. Usually by accident, a slight or even a marked albuminuria may be found in individuals who feel, in general, entirely well, and are, as a rule, still of youthful age. Not infrequently the patients are very strong, healthy-looking children. In other cases the children may be nervous and anæmic, but otherwise they do not give the impression of really being sick. Still, they frequently complain of malaise, headache, general weakness and languor, pains in the limbs, palpitation, anorexia, etc. No cause for this striking symptom can be found, nor are there any other discoverable objective disturbances of the body. If we investigate closely, we find that the urine does not contain albumen constantly, but only at certain times (cyclic or periodic albuminuria). This remarkable variation in regard to the absence and presence of albumen can almost always be traced to variations in bodily rest and activity. When the patient is at rest in bed, or sits quiet, the urine that is voided thereafter (hence every morning urine also) is entirely free from albumen. If the children, however, are out of bed, even for a short time, move about more actively, or perform some gymnastic exercises, a distinct albuminuria develops. This by no means rare form of albuminuria has therefore been given the name orthotic or orthostatic. Sometimes the albumen content of the urine decreases somewhat toward evening. It is worthy of mention that the urine even shows a distinct turbidity on the addition of acetic acid (nucleo-albumen?). Casts (*vide infra*) are usually not present in the urine, but occasionally in a centrifugalized specimen a few hyaline casts are found. The significance of this practically important condition is not quite certain. It is clearly not due to permanent organic changes in the kidney, but may be the result of circulatory disturbances. Possibly, in the children affected, the kidney circulation is interfered with in the standing position, as, for instance, by a kinking of the renal vein. It is claimed that sometimes the unusual mechanical anatomical conditions are indicated by a lumbar lordosis on standing. As has been said, however, the matter has not been entirely cleared up. It is of practical importance to know that in time the condition entirely disappears of its own accord. After the twenty-fifth to the thirtieth

year, orthostatic albuminuria occurs only rarely. The condition is, therefore, as a rule, to be looked upon as not serious, although it must always be regarded as an anomaly, and therefore given a certain amount of careful consideration. Active treatment is unnecessary. Especially is prolonged rest in bed absolutely useless. The children may, in general, lead their customary mode of life. We think that only a certain amount of dietetic precaution is indicated.

Diagnosis.—The detection of albumen in the urine for clinical purposes, wherein no regard need be paid to the separation of serum albumen and serum globulin, is performed almost exclusively by means of the so-called heat test. If the urine is cloudy, it must be filtered before heating. The reaction of the urine must always be tested first. If the reaction of the urine is acid, as it ordnarily is, it is heated in the test-tube without any further addition. If the urine is neutral or alkaline, then, and only then, we acidify it slightly before boiling, by means of a few drops of dilute acetic acid. If the urine contains albumen there will appear, upon boiling, a distinct flocculent precipitate of coagulated albumen. The possibility of an error lies in the fact that sometimes, with a neutral or faintly acid urine, upon heating there appears a cloudiness because of the phosphates and carbonates which are precipitated. These are salts of lime and magnesia. In order to avoid confounding such phosphate deposit with a deposit of albumen, we must in every case after the urine has boiled for a short time, if there is any deposit formed, add a few drops of nitric or acetic acid. By this means the deposit of phosphate or carbonate is immediately dissolved, while a deposit of albumen is not affected. The change in the color of the urine, which is sometimes caused by the addition of nitric acid, is due to the action of the acid upon the urinary pigments. We can measure the amount of albumen contained in the urine approximately by the height of the settled precipitate in the test-tube. We often speak of "one half or one fourth of the volume being albumen," but we cannot state any definite relation between this estimate of the volume and the precise amount of albumen. Approximately, however, a deposit of albumen, which upon settling occupies about half the volume of the urine, corresponds to about one per cent by weight of albumen, a deposit of about one third of the volume to about one half per cent of albumen, and so on. The quantitative estimation of the albumen content by means of the much-used Esbach's apparatus is only of very limited value.

Besides the test by boiling there is a very distinct test which can be highly recommended to practitioners, in which acetic acid and ferrocyanid of potassium are employed. A rather large amount, perhaps one tenth of the volume, of acetic acid is added to the urine, and then a few drops of a ten-per-cent solution of ferrocyanid of potassium are added to the mixture. If the urine contains albumen there is formed a distinct deposit. This reaction usually takes place at once, but if the amount of albumen is very small it may be somewhat delayed.

The so-called Heller's test is also very delicate, but less used. In a test-tube a layer of urine is carefully floated upon concentrated nitric acid. If albumen is present, a distinct cloudy ring will appear at the point of contact of the two liquids.

If we have found out that the urine certainly contains albumen, we must then decide whether we have really a true renal albuminuria—that is, whether

a urine already albuminous is secreted in the kidneys, or whether the albumen is not mixed with a perfectly normal or at least nonalbuminous urine later, in the kidneys themselves or in the urinary passages, the pelvis of the kidney, or the bladder (spurious, accidental albuminuria). Such a spurious albuminuria occurs when the urine is contaminated with blood (as in hemorrhages from the kidneys, the pelvis of the kidney, the bladder, or the urethra), or with pus (in pyelitis, cystitis, etc.). In these cases, of course, the albumen contained in the serum of the blood or pus is found in the urine. Spurious albuminuria is usually easily recognized, since the presence of pus or blood in the urine, which is shown by the appearance of the urine or upon microscopic examination (red blood corpuscles, pus corpuscles), points with immediate certainty to the origin of the albuminuria. Moreover, the amount of albumen in these cases is usually but slight, and corresponds to the amount of pus or blood in the urine. A disproportion in this respect must excite the suspicion whether, besides the spurious albuminuria, there is not perhaps at the same time an affection of the kidneys causing a true renal albuminuria. The determination of this point is not always perfectly easy, but we can usually come to a decision by finding abnormal morphological constituents in the urine, the so-called urinary casts (*vide infra*), which give indubitable evidence of the existence of a disease of the kidneys.

Ætiology and General Pathology of Renal Albuminuria.—What general pathological significance has the true renal albuminuria, and what are the causes of its origin? According to our present theories, the answer to these questions is simply this: In every case of genuine albuminuria there is an abnormal transudation of the albumen of the blood into the urine. Like every glandular organ, the kidney has not only the ability to separate certain substances from the blood, but also the power to prevent the excretion therefrom of certain other substances. Among the latter is serum albumen, and the passage of this from the blood into the urine is therefore directly indicative of an injury to the kidney parenchyma. This change affects both the walls of the blood vessels (analogous to all inflammatory vessel changes) and the epithelium. If the vessel walls become abnormally permeable, and if the epithelium covering also does not hold back the albumen, the latter will enter the secreted urine and be excreted with it. Until now the glomeruli were regarded as the main site of albumen excretion, and disease of the glomerular epithelium was considered the main cause of renal albuminuria. This view, however, is certainly too one-sided. To be sure, we know from experimental investigation that albumen excretion can occur in the glomeruli, but it is quite as certain that it may take place in the convoluted and straight uriniferous tubules. A good example of the excretion of albumen through the glomeruli is furnished by the experimentally produced albuminuria which appears whenever the supply of arterial blood to the kidney is checked by a temporary constriction of the renal artery. The epithelium of the glomeruli thereby suffers a visible microscopic change. If the kidneys in this condition are removed as rapidly as possible and boiled, according to Posner's suggestion, we can discover under the microscope in the capsules of the glomeruli the albumen that is thus coagulated (Ribbert)—a most certain sign that the passage of the albumen from the blood vessels into the urinary passages has in fact taken place in the glomeruli. On the other hand, we know from the

experiments of Senator that, in artificial venous hyperæmia, the epithelium of the uriniferous tubules suffers earlier than that of the glomeruli. Finally, we have the toxic nephritis following vinylamine poisoning, that has been studied by Ehrlich and A. Heinecke, in which the cortex of the kidney remains almost entirely intact, while the medullary pyramids become intensely diseased. Here, also, most pronounced albuminuria develops. It follows that in the various pathological processes the site of the principal albumen excretion is probably not always the same. We must still believe, however, that, at least in many cases, especially in the many milder and rapidly disappearing albuminurias the glomerulus is the chief site of the albumen excretion.

Compared to the changes in the kidney tissue, including its blood vessels, all other factors no doubt play only a secondary rôle in the causation of albuminuria, although they may, of course, have an influence upon the quantity of albumen excreted.

The changes in the composition of the blood, on which formerly, and again by some investigators recently, great stress has been laid, especially the hydræmia and hypalbuminosis (the diminished amount of albumen) of the blood, have probably only an indirect significance, since the nutrition of the walls of the glomeruli suffers from such a faulty condition of the blood, and this circumstance again influences the true cause of elimination of the albumen. The significance of the blood pressure with regard to the occurrence of albuminuria was also formerly very much overrated. According to the older hypothesis, it was believed that, in an increase of the blood pressure, the molecules of albumen in the blood could be pressed through the filter formed by the membrane of the glomeruli. This hypothesis has been disproved, especially by the experiments of Runeberg; these experiments showed that, in the filtration of solutions of albumen through animal membranes, a rise in the filtration pressure was followed by a decrease, and a fall in the pressure by an increase of percentage of albumen in the filtrate. The extent of the albumen excretion is frequently and distinctly affected by circulatory disturbances. If the condition of the kidney epithelium suffers from the circulatory disturbances, albuminuria frequently develops or is increased (*vide infra,* chapter on Congested Kidney).

2. CASTS AND OTHER ABNORMAL MORPHOLOGICAL CONSTITUENTS OF THE URINE IN RENAL DISEASE

Besides albuminuria, certain peculiar morphological constituents of the urine, visible under the microscope, are of especial importance for the diagnosis of renal affections, namely, the urinary casts, whose significance was first correctly recognized by Henle in 1842. These are cylindrical bodies, whose breadth corresponds to the width of the uriniferous tubule, and whose length only exceptionally reaches a millimeter, which must be regarded in their chemical nature as consisting mainly of a coagulated albuminous substance. To the latter circumstance we owe their old name of " fibrin casts," or " fibrous casts," a name which is obsolete, and properly so, since the coagulated albuminous substance of casts is certainly not identical with fibrin.

Since the precise conditions of the occurrence and the character of the

renal casts, in the different diseases of the kidneys, will be spoken of later, we need discuss here only the general properties, the origin and the significance of casts (see Fig. 95).

1. *Hyaline Casts.*—The hyaline casts are the commonest and most important form of casts, and, to a certain extent, are the ground form for different varieties. They are perfectly homogeneous, clear as glass, colorless, soft, and flexible. We find them either wide or narrow, sometimes broken off short, sometimes quite long, usually straight, but in many cases partly curved. They are easily stained with carmine or gentian violet. On heating the urine they are dissolved, but they are quite resistant to acids.

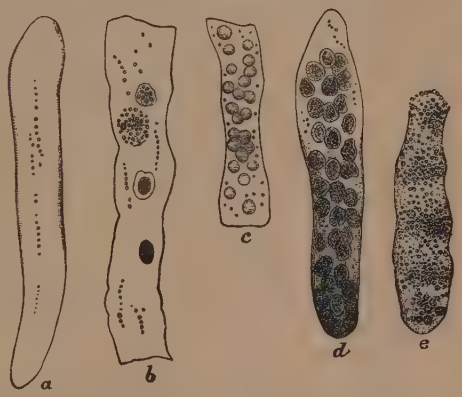


FIG. 95.—Different forms of casts. *a.* Hyaline cast with occasional granules. *b.* Hyaline cast with fat drops and granular cells. *c.* Hyaline cast with red blood corpuscles attached. *d.* Hyaline cast with white blood corpuscles attached. *e.* Cast with a large number of fat drops.

The hyaline casts are very often covered to a greater or less extent with all sorts of deposits, which are usually affixed to the soft substance of the cast in the kidney itself, but which may often be attached to it later. These deposits are as follows: First, red blood corpuscles. This condition is important, because it points with certainty to the existence of hemorrhages in the kidneys themselves. Second, of white blood corpuscles. These are often considerably swollen, so that we must guard against mistaking them for epithelium. Third, of renal epithelium, which may be recognized by its size, its more angular shape, and its nuclei. Of course we often find the epithelium cloudy and granular, or shriveled and atrophied. Fourth, of fatty granular globules —that is, both fatty-degenerated epithelium and also white blood corpuscles which are filled with fat drops from the fatty-degenerated cells. Fifth, of little granular masses whose nature cannot always be easily recognized. They are either coagulated granules of albumen, or fat drops, or urates, or bacteria, or, finally, granules of hematoidin, which have come from the destruction of red blood corpuscles, and are usually easily recognized by their dark, brownish-yellow color. Sixth, we rarely find in the casts drops like myelin, as to whose precise significance nothing is known.

As to the origin of hyaline casts, despite many investigations the question has not been fully answered. Probably they are due to the coagulation of the excreted albumen occasioned by the dying epithelial cells and the escaped leucocytes.

Two factors are, therefore, always necessary for the formation of casts, viz., the excretion of coagulable albumen and the presence of substances to produce coagulation. In this way the fact is explained that we sometimes have a marked albuminuria with only very slight cast formation. When, on the other hand, we observe casts in the urine without albuminuria, the casts are usually not of the ordinary hyaline variety, but are some other formation in

the uriniferous tubules. Besides, the number of casts found in the urine does not always correspond to the extent of their formation. In the severest forms of acute nephritis almost all the tubules can be plugged with casts which may be washed out into the urine to only a very slight degree.

2. *Epithelial Casts.*—The epithelial casts are composed exclusively of renal epithelium, although probably hyaline casts not infrequently form a basis to which the epithelial cells adhere. Epithelial casts are usually easily recognized, and always indicate an excessive desquamation of epithelium in the diseased kidneys. One must be on one's guard, as already mentioned, not to confound renal epithelium with swollen white blood corpuscles. The separate epithelial cells and the epithelial casts may present various changes, such as granular opacity, fatty degeneration, and atrophy.

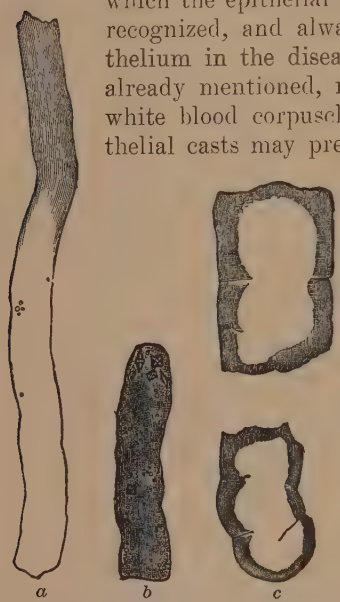


FIG. 96.—*a*. Waxy cast. *b*. Waxy cast bearing crystals of calcic oxalate. *c*. Fragments of waxy casts. (From von Jaksch.)

3. *Waxy Casts.*—The so-called waxy casts are almost always rather broad and usually yellowish colored, opaque casts, of evidently a much tougher consistence than hyaline casts. We have found them most frequently in severe acute nephritis, either primary or secondary to scarlet fever, but they also appear in the severer forms of chronic diffuse nephritis. As to their origin, we are convinced that they certainly in many cases are formed out of epithelial casts. The closely apposed renal epithelial cells degenerate into waxy flakes, and gradually coalesce. One can, in fact, observe all stages of transition between the epithelial casts and the almost completely homogeneous waxy casts. Fürbringer, therefore, terms waxy casts "metamorphosed," and he insists, correctly, that they always indicate severe disease of the kidney.

FIG. 97.—Epithelial cast, granular in its lower half. (From von Jaksch.)

4. *Granular Casts.*—A kind of cast which is composed of coarse, yellowish, flaky granules represents the just-mentioned transition of epithelial casts into waxy casts. In other cases the waxy casts are nothing but hyaline casts completely covered with the above-mentioned granules of various kinds. Sometimes, also, coagulated masses of albumen or granules of hematoidin may themselves be formed into cylindrical shapes.

Genuine blood casts are not very frequent. They consist of coagulated blood and represent casts of the renal canals into which hemorrhage has taken place.

Casts in Diagnosis.—The clinical diagnostic significance of renal casts is very great. They are, in the first place, always a sure sign of the existence of some renal disease, since in normal urine casts are not found at all, or, at most, they are exceptional and are present in small numbers. The consider-

ation of the special forms of casts, and of the deposit upon them, is also of great diagnostic importance, although from it we can never decide immediately upon the general form of the renal disease, but we can recognize with certainty the type of special pathological processes in the kidneys. Frerichs has well named casts "messengers from the kidneys." The blood casts and the red blood corpuscles sticking to the cylinders point to the occurrence of renal hemorrhages; the epithelial casts to a desquamation of the epithelium in the kidneys; the white blood corpuscles to an emigration of the colorless cells from the vessels; the fatty granular cells to processes of fatty degeneration in the kidneys.

We have already learned to recognize in a great measure in the preceding, as occasional deposits on the casts, the other morphological constituents found, besides the casts, in the sediment of the urine in renal disease. Briefly recapitulated, they are as follows:

1. *Red blood corpuscles.* The presence of a large amount of blood in the urine (hematuria) is almost always to be recognized by its blood-red color. The blood may be made out with certainty by the microscope, or by Heller's blood test. The latter is performed by heating the urine in a test-tube with sodic or potassic hydrate. The blood corpuscles are thus dissolved, and the hematin formed from the blood pigment is precipitated with the phosphates, giving to the precipitate of the latter a very characeristic blood-red color. Van Deen's test is very distinct and easily performed. It requires a freshly prepared mixture of equal parts of old oil of turpentine and newly made tincture of guaiac. This mixture is poured upon the urine to be examined, and slightly shaken. At the place of contact of the two fluids there will immediately form a beautiful blue ring, if there is the slightest trace of blood in the urine. It is important to know, however, that urine containing pus also reacts positively with the guaiac test. Finally, of course, the spectroscope may serve for the detection of hematuria. Hemoglobinuria will be described in a special chapter later.

2. *White blood corpuscles.* Only when they are also attached to the casts can we assume with certainty that these come from the kidneys, and not from the lower portions of the urinary tract.

3. *Renal epithelium.*

4. *Fat drops and fatty granular cells.*

5. *Uric-acid crystals, urates and calcic oxalate, bacteria,* etc.

For microscopic examination of the urinary sediment, we let the urine settle in a tall beaker; it is more convenient, and, if the urine contains few morphological constituents, far more reliable to employ a centrifugal machine.

3. THE DROPSY OF RENAL DISEASE

Although the changes in the urine must be alone decisive in the diagnosis of any renal disease, there are yet certain other symptoms which are also due immediately to the renal affection, and which may first direct our suspicions to the existence of a disease of the kidneys, and consequently lead to a careful examination of the urine. Among these symptoms the dropsy of renal disease is one of the commonest and most important. Every trace of œdema may, indeed, quite frequently be entirely absent, both in acute and chronic nephritis,

and in other diseases of the kidneys; but in many cases the dropsy is decidedly prominent in the whole clinical picture.

Ætiology and Pathology.—If we ask what is the reason of the frequent occurrence of dropsy in renal disease, the answer at first does not seem difficult. Since the main function of the kidneys is to excrete water from the body, and since, as we shall see later, in many cases the diseased kidney can no longer fulfill this task, or can fulfill it only to a slight degree, we are not, in fact, very much out of the way in considering the retention of water in the body as the main cause of the consequent œdema. Clinical observation seems in general to agree completely with this assumption. The œdema in renal disease seldom appears until the daily amount of urine has been below the normal for some time, while, on the other hand, in those cases where the amount of urine passed is normal, or even abnormally great, in spite of the existing renal disease, œdema is wholly absent. In individual cases, too, we very often see a decrease of the œdema associated with an increase in the amount of urine, and an increase of the œdema associated with a corresponding diminution in the excretion of urine. The pathological process accordingly seems to consist of an accumulation in the body of the water which cannot be excreted from it, and which transudes from the vessels and thus gives rise to the development of œdema.

On more careful consideration, however, there are some objections to this theory, which is apparently so simple. In the first place, it might be supposed that, when there is retention of water, the body must get rid of the surplus water by employing to a greater degree the other channels of elimination which are at its service—the skin and the intestines. Since we can never determine accurately the time when the water first begins to be retained in the body, the clinical experience just mentioned may also be thus interpreted, that the lessened excretion of urine is not the cause of the œdema, but that, on the contrary, the appearance of œdema is rather the cause of the diminished elimination of water by the kidneys. The other clinical facts, also, are inconsistent with the view that the œdema depends upon the simple accumulation of water in the blood. In many forms of nephritis, and particularly in the nephritis of scarlet fever, we very often see extreme œdema appear suddenly, while, on the other hand, many severe varieties of nephritis—e. g., those associated with diphtheria, septic diseases, and pneumonia, run their course with little or no œdema, although they are characterized by a marked diminution in the amount of urine. Moreover, cases have been repeatedly observed in which, as a result of obstruction of the ureters, or from pressure on these canals, there has been complete anuria for several days without a trace of œdema developing; and experimental investigations have given corresponding results. The bilateral ligation of the ureters in animals does not lead to the development of œdema even after several days. Cohnheim and Lichtheim introduced large amounts of a one-half to one-per-cent solution of common salt into the vascular system of an animal, and despite this great artificial "hydræmic plethora," observed no œdema, even when the renal arteries were tied. Nor can œdema be provoked by slow and continuous infusion (Gärtner, Francotte).

Consequently, we must seek some other cause to explain the œdema of nephritis, and this is in all probability the peculiar change in the vascular

walls, which renders them more permeable and permits the water accumulated in the blood to transude into the tissues. Just what this change in the vascular walls may be and what produces it we do not yet know; probably there are chemical agents which change the vascular walls, either the same substances which excite the nephritis, or matters secondarily formed, or certain products of tissue metabolism retained in the blood because of the inefficiency of the kidneys. This explains why certain forms of nephritis produce pronounced œdema and others do not. In experimental nephritis, the occurrence of œdema is also dependent upon special circumstances. Certain kidney poisons (chromium salts, cantharides, etc.) always produce a nephritis without œdema, while, on the other hand, the nephritis produced by uranium salts is almost always associated with pronounced œdema. In the blood serum of animals poisoned with uranium a substance is present that causes œdema. If a chromium nephritis is produced in an animal, and this animal is injected with blood serum, or œdematous fluid of an animal suffering from an uranium nephritis, an œdema will follow (A. Heineke). The rôle played by changes in the osmotic tension of the blood and tissues in the development of œdema is not yet perfectly clear. Especial weight has been laid on the not infrequently observed diminution of the sodium chlorid excretion (H. Strauss) in nephritics. The consequent increase of the sodium chlorid in the blood serum may lead to an increased passage of that salt into the tissues, with a corresponding exudation of water.

That dropsy develops because of direct damage to the blood vessels is rendered probable from the cases of so-called "acute essential dropsy," in which there develops a considerable general dropsy, just as in acute nephritis, often preceded by mild gastrointestinal symptoms, and yet without a trace of albumen in the urine or any other demonstrable cause for this dropsy, which usually soon abates.

Many clinical peculiarities of nephritic œdema harmonize with this conception of its origin. In general, we may say that the œdema of nephritic patients appears first in the skin ("anasarca"), and has a striking tendency to show first in the face, in contrast with the œdema due to the passive congestion of heart disease, which usually begins in the ankles. The puffy and noticeably pale countenance of the patient will often suggest the existence of kidney trouble at the first glance. In severe cases the dropsy often develops over the whole body in an extreme degree, involving the skin of the trunk, and particularly the dependent parts, the extremities and scrotum, so that the patient is a pitiable object. In such cases there are also apt to be dropsical transudations into the internal cavities of the body, causing hydrothorax, ascites, and hydropericardium; and these aggravate the patient's distress. It is, however, a striking fact that often there will be marked dropsical effusions (ascites, hydrothorax), and yet no marked œdema of the skin; and hydrothorax is sometimes much greater on one side than on the other. All these facts indicate special local conditions—viz., the changes in the blood vessels, which we have surmised. Still more is this the case with regard to the œdema of mucous membranes which sometimes occurs. There may be œdema of the conjunctiva, of the soft palate, of the aryepiglottic ligaments (œdema of the glottis), and other parts. Œdema of this sort is apt to resemble a mild local inflammation, and in general it is impossible to deny a certain relation-

ship between "inflammatory" œdema and nephritic œdema. The nephritic œdema of the skin may in places have suggestions of an inflammatory nature, such as slight redness and tenderness. Œdema may affect the internal organs, and nephritic pulmonary œdema is of great practical importance. The question of the existence of cerebral œdema and of its possible importance will be discussed below (see uræmia).

The preceding remarks relate only to genuine nephritic œdema. We shall see later that œdema may have an entirely different origin, particularly in cases of chronic nephritis—that is, if there is a cardiac hypertrophy and the powers of the heart become impaired, dropsy may at last develop. This œdema is, of course, a genuine congestive œdema, and exactly similar to that seen in failing compensation in heart disease.

In its chemical composition, the dropsical fluid corresponds to a very thin blood serum. The amount of water is usually ninety-seven to ninety-eight per cent. The amount of albumen is usually very slight, while the percentage of sodium chlorid (one to one and a half per cent) is always somewhat higher than in the blood serum. This fact indicates an active participation of the vessel walls in the formation of the œdema. The high sodium-chlorid percentage of the œdema fluid is connected with the disability of the diseased kidney to excrete all of that salt which is brought to it (*vide infra*). In this way the body maintains the necessary normal sodium-chlorid content of its blood. An increased intake can therefore under certain conditions lead to an increase of the œdema.

4. URÆMIA

If the diseased kidneys can no longer perform their secretory functions in a satisfactory way, not only does the elimination of water from the body thereby suffer, but the soluble constituents of the urine, the salts, the urea, and the other final products of tissue metamorphosis may also be retained in the blood and accumulate there. Hence we often find the blood, in patients with renal disease, not only more watery than under normal conditions, so that the specific gravity of the serum may fall from 1.030 to 1.020, or even lower, but, in almost all cases where there is a diminished excretion of urine, it is also richer in urea, as many experiments have shown, and under corresponding conditions it is probably also frequently richer in the other constituents of the urine, or in substances corresponding to them, but not completely metamorphosed.

This accumulation of the urinary constituents in the blood and, further, perhaps in the tissues themselves, is the cause of a class of symptoms which are often seen in diseases of the kidneys, and which are termed uræmic symptoms or uræmia.

Ætiology and Pathology.—Probably no one to-day doubts that uræmia must be regarded as essentially an intoxication of the body by the retained products of tissue metamorphosis. Numerous experimental investigations have proved that in animals extirpation of the kidneys, or ligation of the ureters, will produce a symptom-complex, characterized by vomiting, convulsions, and coma, completely analogous to the uræmia of Bright's disease; but if we inquire what constituents of the urine are the particular occasion of the uræmic phenomena, we cannot as yet obtain any definite answer. For a long time it was believed that urea played a chief part in the development

of uræmia, but the result of experiments upon animals does not support this view. It is possible to inject enormous amounts of urea into the circulation or into the peritoneal cavity of animals without any symptoms of poisoning. Voit did, indeed, show that the healthy kidneys remove from the blood the excessive amount of urea with extreme rapidity, and that accordingly uræmic symptoms do really appear if, while we are feeding an animal with large amounts of urea, we impede the excretion of the urea by a simultaneous withholding of water. Yet the amount of urea necessary for the success of this experiment is greater than can possibly exist in the ordinary uræmia of Bright's disease, and, moreover, the withholding of water might also prevent the excretion of other matters. The hypothesis of Frerichs, accepted in many quarters for a time, has also been universally discarded. He assumed that through fermentation the urea in the blood of nephritics was converted into carbonate of ammonia. Hence we must seek for other poisonous substances as factors in the production of uræmia. Many experiments seem to indicate that the potassium salts are poisonous, while some authors have laid the blame mainly on the extractive matters, such as creatinin. Bouchard has sought to prove that certain alkaloid substances (urotoxins), which are presumably developed during the digestion of albumens and are always demonstrable in normal urine, occasion the phenomena of uræmia, while other authors (Ascoli and others) speak of special poisons, the product of disintegration of the tissues ("nephrolysin," "nephrotoxin"). At any rate the fact is worthy of note that the suppression of the urinary excretion in kidneys that are otherwise healthy—for instance, in obstruction of the ureter by an impacted calculus—may be tolerated many days without the occurrence of uræmia. It would thus seem that there are still certain factors in the kidney disease, as such, which must be considered in the development of the uræmic phenomena. In accordance with certain modern views regarding the significance of the so-called internal secretions, some investigators also assume an "internal secretion" from the kidneys into the blood, and believe that the uræmia can be traced to a disturbance of this function somewhat analogous to similar occurrences in diseases of the liver, the thyroid gland, and the suprarenals. But against all of these hypotheses objections may be offered, so that the question of the more exact nature of the products which occasion uræmic intoxication must still be regarded as unsolved. The newer physico-chemical examinations of the blood have likewise given no certain solution. The molecular concentration of the blood is naturally usually increased without, however, the existence of a regular parallelism between the severity of the uræmic symptoms and the degree of the retention. The electric conductivity of the blood is not increased in uræmia, which would indicate that the inorganic blood salts do not play any causative rôle. From the chemical standpoint, an increase in the so-called residual nitrogen (i. e., the nitrogen remaining in the blood after the complete precipitation of albumen) is of significance, as from this we might suspect that it is the nitrogenous decomposition products of albumen which produce the uræmic poisoning. It is not impossible that, after all, a number of substances come into play.

The mode of action of the toxic material is as unsettled as is its chemical nature. This much only is certain, that in uræmia the disturbances are almost exclusively cerebral, and in the main located in the cortex of the brain; but

whether there is actually a direct injury of the nervous elements, or whether the immediate action is upon the blood vessels (spasm), is uncertain. The occurrence of uræmic focal symptoms, to which we shall refer below, renders the former supposition the more likely.

Clinical experience agrees perfectly with the theory that uræmia is caused by a retention of urinary constituents in the body. In most cases the uræmic symptoms appear only when the daily amount of urine has fallen to a very low figure, or when the secretion of urine has wholly ceased for several days. That in these cases not only the elimination of water, but also the elimination of the other urinary constituents, is very much diminished, is shown by experiments made in regard to this point.

Of course there can be no question that some clinical facts cannot be brought into exact harmony with what has been previously said. If cases are repeatedly reported in which no uræmic symptoms have appeared in spite of anuria lasting several days, it does not prove very much, since we can never make an exact estimate of the matter accumulated in the blood which ought to have been eliminated; for the organism can certainly get rid of the final products of tissue metamorphosis in other ways than through the kidneys—for instance, through the skin, the intestines, or the fluid of œdema—and we must also bear in mind that different individuals show a great diversity in tolerating the action of any poison in the body, particularly as regards the nervous system. It is harder to explain those cases which are sometimes seen, in which uræmic symptoms suddenly appear in patients with renal disease, although these symptoms are not preceded by any noticeable diminution of the secretion of urine. We may, however, assume that, despite the abundant excretion of water and the normal amount of urine, there has been a slight retention of solid matters. Analogy with other kinds of poisoning makes the supposition very plausible that the long-persistent retention of even extremely small portions of toxic matter may occasion an absolutely sudden explosion of the severest symptoms. In chronic lead and mercurial poisoning the symptoms often appear with great abruptness, although the poisoning has taken place very slowly and gradually. In the same way we explain to ourselves the not very infrequent cases of the sudden development of severe uræmic symptoms, as seen particularly in patients with contracted kidney (*vide infra*), in whom it may seem as if no single prodromal symptoms had indicated the impending outburst of intoxication. Often, also, peculiar circumstances may favor the occurrence of uræmia; for example, the development of cardiac weakness, so that the blood tension is diminished and the excretion of urine impeded. In some cases again it is observed that the development of uræmia coincides with the absorption of previously existing œdema. This is explained by the supposition that the rapid absorption of the œdema introduces into the blood a comparatively large amount of the poisonous products of metabolism, which had not been excreted, but had been contained in the œdematous fluid.

While we are firmly of the opinion that uræmia is to be regarded as a poisoning of the body by the retained constituents of the urine, yet we should not fail to mention that attempts have been made to explain in other ways the uræmic phenomena. In particular, Traube has propounded the theory that the so-called uræmic symptoms are dependent upon an acutely developing cerebral œdema, and consequent cerebral anæmia. This theory can be regarded

as, on the whole, untenable. Still, it may contain a certain amount of truth, for we cannot wholly deny that sometimes there may be actual anatomical lesions, such as inflammatory œdema of the brain, in the course of a nephritis, sufficient to occasion severe cerebral symptoms. During nephritis secondary inflammation may appear in almost all the internal organs, and often suddenly; and this sort of nephritic inflammation is especially frequent in the retina, a structure composed of nervous elements, so that the possibility of similar disease in the brain is very great. If we consider that cases of uræmic hemiplegia and monoplegia have been repeatedly observed, and that likewise cases have been described of uræmic Jacksonian epilepsy, hemianopsia, and aphasia, it will be seen that the supposition of an actual local lesion as the explanation of such well-marked focal symptoms is very plausible. Yet we must also consider that in the last analysis the cause of such limited œdematous—or inflammatory-œdematous—changes in the brain is to be sought in the action of some retained products of metamorphosis.

Symptoms.—In regard to the clinical symptoms of uræmia in the individual case, they show all possible transitions, from the mildest symptoms, which are only intimated, up to the severest nervous symptoms, which may be the immediate cause of death. The severe forms of uræmia may sometimes come on quite suddenly, while in other cases they may be preceded for a long time by milder uræmic symptoms, which are then termed prodromata. The severest symptoms may not appear at all, and the milder symptoms may exist alone for a longer or shorter time. This latter condition is called chronic uræmia.

The milder uræmic symptoms, which are observed either alone or as precursors or as sequelæ of severe uræmia, consist of headache, somnolence, and mental stupor; of a peculiar uneasiness, or of a feeling of anxiety and constraint (sometimes associated with hurried respiration), and very often of nausea, spasmodic eructations, and repeated vomiting (*vide infra*). Very characteristic of uræmic intoxication are signs of motor irritation, especially isolated, brief twitchings, or even persistent tonic rigidity of the extremities or face. Signs of sensory irritation, formication and numbness of the fingers, and also particularly an annoying, persistent itching of the skin (*vide infra*) are not uncommon. Among the symptoms of this milder form of uræmia, besides the vomiting, we would place the peculiar restlessness and præcordial distress of patients as especially important. Not infrequently observers use the term uræmic asthma (*vide infra*).

The most characteristic symptom of severe uræmia is the uræmic convulsion. It corresponds almost exactly in its details to an epileptic attack; it usually begins with a short tonic stage, in which the whole body is generally in a position of extension in opisthotonos, and then follow vigorous clonic contractions in the face and extremities. The face becomes cyanotic, a bloody froth comes from the mouth, the pupils are usually dilated and almost without reaction, the respiration is accelerated (but at times it is intermittent from spasm of the respiratory muscles), the pulse is small and accelerated, and it can scarcely be felt in the radial artery, and the temperature is sometimes raised. In other cases the spasm begins with short, jerky contractions in one extremity, as in the arm, and then invades the trunk muscles, the face, and the legs. One half the body is often more affected in the attacks than the

other. The spasms usually cease in a few minutes, and are followed by deep coma and stertor, or even Cheyne-Stokes' breathing, which last for several hours or more. There is only rarely a single attack. The attacks are much oftener repeated after longer or shorter intervals, so that there may even be twenty or more in the twenty-four hours, during the whole of which time a complete loss of consciousness persists. Severe and fully developed epileptiform attacks often alternate with slighter convulsions.

Some other uræmic symptoms, besides the convulsions, which have already been briefly mentioned, merit a somewhat fuller description.

The uræmic amaurosis occasionally seen is especially interesting. It is usually left after recovery from the convulsions. Only rarely does it precede them or appear without them. It always develops quite rapidly, so that the first disturbance of vision soon passes into complete blindness. The reaction of the pupils to light is almost always retained, and the ophthalmoscope shows a perfectly normal retinal image. At what spot in the visual apparatus the lesion is situated is not yet known. Many investigators assume that there is an œdema of the sheath of the optic nerve; while others, including the author, think it more probable that there is disturbance of the cerebral visual centers, and particularly of the occipital cortex.

Its prognosis is, on the whole, favorable, since the disturbance of vision usually disappears completely in a day or two, though sometimes not until after a longer time. Anomalies are only rarely seen in the domain of the other nerves of special sense, the most frequent, comparatively, being a difficulty in hearing, or even complete deafness.

Other motor disturbances, except twitchings and convulsions, are rare. Only in a few cases have hemiplegic or monoplegic paralyses, contractures, trembling, etc., been observed. Mental symptoms are more common. Delirium, and maniacal or sometimes melancholic states, occasionally follow uræmic coma.

Those uræmic symptoms also have a great interest which are to be regarded as a sort of self-help on the part of the organism, since they often lead to a vicarious elimination of urea and presumably of the other products of tissue metamorphosis. The first of these is uræmic vomiting, which is a frequent and often an extremely obstinate symptom both in acute and chronic uræmia. In many cases it is of central origin, and is to be regarded as analogous to the vomiting so frequent in different forms of cerebral disease; but it is often produced by the irritation which the gastric mucous membrane suffers from the urea eliminated, or rather from the carbonate of ammonium arising from it. The latter is always first formed from the urea in the stomach itself, and we find in the vomitus of uræmic patients either the still undecomposed urea or the carbonate of ammonium in considerable quantities. Sometimes there is quite a violent hiccough besides the vomiting.

Uræmic diarrhea has the same significance as uræmic vomiting. It is usually provoked by the carbonate of ammonium arising from the urea in the intestines. The former often causes quite a severe catarrhal, and even at times a diphtheritic, inflammation of the intestinal mucous membrane.

Another way in which the organism sometimes tries to get rid of the large amount of urea accumulated in it is by the sweat glands. Schottin first described the remarkable discovery of a coating of urea on the skin in the

uræmia of cholera, an observation which since then has been repeatedly confirmed in other cases of uræmia. This coating is most frequently seen on the face, especially on the sides of the nose, to which little faintly lustrous scales are seen sticking after the evaporation of a clammy sweat. Chemical examination shows that these scales are urea. The excretion of urea is much more rare in other parts of the skin, but perhaps the occasional severe uræmic itching of the skin is due to an irritation of the cutaneous nerves by some of the constituents of the urine that are excreted.

Other organs besides the skin and the digestive tract are but rarely to be considered as a means of the vicarious elimination of urea, but Fleischer was once able to discover considerable amounts of urea in the saliva and sputum of a uræmic patient.

In conclusion, we must describe the condition of the pulse, of the temperature, and of the respiration in uræmia. The pulse is often very slow before the appearance of severe symptoms, sometimes 48 or 40, but it is almost always tense and hard. In chronic uræmia, also, a moderate slowness of the pulse is not infrequent. When uræmic convulsions appear, however, the pulse usually becomes small and very frequent, especially in cases that terminate unfavorably. The temperature but rarely remains unchanged in severe uræmia. If there are convulsions, it usually rises several degrees, in severe cases even to 106° or 108° F. (41° to 42° C.). We have seen these high temperatures, especially, as a terminal rise with an unfavorable issue, although there may sometimes be an improvement even in such cases. On the other hand, there are also great declines in temperature, down to 93° or 91° F. (34° to 33° C.), most frequently again as a terminal temperature of collapse, in cases which end in deep coma without marked symptoms of motor irritation. We might also mention the "uræmic chills" which we have seen several times—that is, a chill coming on suddenly along with other uræmic symptoms, with a great increase of temperature, and followed by a rapid fall in the temperature. The respiration in uræmic patients is sometimes very much accelerated, and is especially deep—a symptom which recalls the peculiar breathing in diabetic coma (*vide infra*). Certain severe attacks of dyspnœa in patients with renal disease have been described as "uræmic dyspnœa" or "uræmic asthma," but it is not always easy to decide whether this is really a uræmic nervous symptom in these cases, since similar conditions of sudden dyspnœa may depend upon coincident heart disease and insufficiency of the left ventricle, or upon inflammatory affections of the lungs. The occurrence of Cheyne-Stokes' breathing in uræmia has already been referred to above.

In regard to the duration of uræmic symptoms and to the different forms and ways in which the various uræmic symptoms may be combined in the clinical picture, we can give only a few general statements. The division of uræmia into an acute and a chronic form, already mentioned, is generally very useful practically. In the acute form we usually have the severe uræmic symptoms, especially uræmic convulsions and uræmic coma. This condition usually lasts some days, while chronic uræmia, in which the milder cerebral symptoms—uræmic vomiting, difficulty in breathing, etc.—are most prominent, may last as many weeks. The severe acute form of uræmia may, as has been already stated, begin with almost absolute abruptness. Quite often, however, there are first mild uræmic symptoms, such as headache, vomiting,

general restlessness, and an occasional slight twitching of the muscles, and then suddenly uræmic convulsions or other uræmic symptoms.

Prognosis.—The termination of uræmia is always doubtful in every severe case, but it is by no means always unfavorable. Even after coma lasting for several days, with very severe and often repeated convulsions, the uræmic symptoms may wholly disappear, while on the other hand, of course, uræmia is by no means a rare cause of death in the most diverse forms of acute and chronic renal disease. In judging of the individual case, the most stress is to be laid on the condition of the pulse, the respiration, and the temperature; we must also consider, of course, the character of the urinary secretion, and especially the other morbid symptoms dependent upon the primary disease.

5. THE CHANGES IN THE CIRCULATORY APPARATUS IN RENAL DISEASE

Although it had not escaped Bright's observation that changes in the heart are also present in diseases of the kidney, this condition was first generally known when Traube, in 1856, in a treatise which has become famous, explained that a change in the heart was very common in certain renal affections, and thus gave the chief impulse to the numerous clinical and experimental investigations that have been made since then as to the connection between cardiac and renal disease.

This connection, generally considered, may be accounted for in three ways:

First, the heart disease may without doubt be the primary disease, and only secondarily lead to a disease of the kidneys. In this way develop the kidney of passive congestion (*vide infra,* and page 433), acute nephritis secondary to acute primary or recurrent endocarditis, and the embolic processes in the kidney (*vide infra*).

Secondly, heart disease and renal affections may also develop independently of each other, as a result of an injurious influence that affects both organs at the same time. Thus, for example, a general arteriosclerosis leads to cardiac hypertrophy or to myocarditis, and also to a granular kidney (*vide infra*), as a result of an implication of the renal vessels. Certain other injurious influences, such as toxic and constitutional influences, alcohol, syphilis, or improper living, may also cause a disease of the heart and the kidneys at the same time. Later on, if both affections have developed, their influence upon each other is often, of course, considerable—a circumstance which may render our judgment as to the condition decidedly difficult.

In the third place—and this is the point with which we are here chiefly concerned—the renal affection may be the primary disease, and of itself the cause of a change in the heart. This is usually a secondary hypertrophy of the heart, particularly of the left ventricle, but also of the other parts of the heart. There can be no doubt of the fact of this dependence. We also know that the secondary development of cardiac hypertrophy is not confined to one form of chronic nephritis, the so-called contracted kidney, as was at first believed, but that it is just as constant in all other forms of chronic, and also even in prolonged acute, nephritis. Opinions are at present still much divided as to the precise nature of this connection, and as to the causal factors.

The theory which Traube himself advanced for the explanation of the cardiac hypertrophy in nephritis rested on the assumptions that, in the first place, less water is withdrawn from the blood in nephritis for the formation of the renal secretion, and that, in the second place, the flow of arterial blood into the venous system is hindered by the changes in the kidneys, particularly by the destruction of many of the smaller blood vessels. Both circumstances must raise the pressure in the arterial system, and therefore gradually lead to cardiac hypertrophy. Traube's theory cannot be maintained. The first claim especially is untenable, because in many cases of chronic contraction of the kidneys with coexisting cardiac hypertrophy there is never a diminution of the elimination of water by the kidneys, and, besides, this can never of itself cause an increase of the arterial pressure. The second supposition, that the destruction or the narrowing of a large number of the small blood vessels of the kidneys must produce a general rise in arterial tension, is disproved by the fact that even the complete ligation of both renal arteries does not raise the tension in the arterial system, because the blood at once passes off into other vessels which dilate.

In place of the "mechanical theory," therefore, there have been of late many supporters of the "chemical theory" of cardiac hypertrophy, which was in a certain sense propounded by Bright himself, and later by Johnson and others. According to this view which, with some modifications, is held by the author, the retention of urinary constituents, or other abnormal material, in the blood is the cause of the cardiac hypertrophy because the retained material occasions a rise in arterial tension, and this increased tension, if it persists for a sufficiently long time, must occasion an hypertrophy of the left ventricle. Clinical experience shows beyond a doubt that any severe case of acute nephritis occasions in a few days an increase in the arterial tension, which can usually be easily perceived in the pulse. This increased tension, which certainly precedes the cardiac hypertrophy, is best explained by the supposition that the matters which ought to be but are not excreted occasion a contraction of the smaller arteries. It is also possible to conceive that there may be a direct irritation of the cardiac muscle. At any rate, the occurrence of a rise in arterial tension is of decided benefit to the body, as it promotes the excretion of urine. If, in the course of a nephritis, the normal conditions return after a few days or weeks, the increased tension is relaxed and the heart suffers no noticeable change; but if the nephritis and consequent impairment in urinary secretion and increased arterial pressure persist for a considerable time, we often see, even after six or eight weeks, a hypertrophy of the left ventricle develop, under our own eyes, in a way that can be demonstrated most distinctly at the bedside. This is the necessary consequence of the increased effort which the heart is obliged to make in order to overcome the abnormal resistance in the systemic arteries.

We have precisely the same conditions in the chronic forms of nephritis, except that they develop more slowly and insidiously. In these, also, the first factor is the insufficiency of the kidneys, occasioned by the disease—that is, failure to excrete all the products of metabolism. To this the body responds immediately by a rise in arterial tension, which is intended to serve, and does actually serve, as a compensation of the impairment; then, thirdly, hypertrophy of the left ventricle develops, and enables the heart for a long while

to supply the demands put upon it. This hypertrophy is consequently the most important compensatory arrangement by means of which the body is protected from the onset of uræmic intoxication. Just as any patient with valvular heart disease would invariably fail much earlier if his heart did not become hypertrophied in portions corresponding to the lesion, so, also, in chronic nephritis, the unfavorable termination would occur much earlier if the body were not in a position to effect and maintain an increase in the arterial tension, and thus for a time, at least, ward off the threatening enemy. Viewed in this light, the chemical theory of cardiac hypertrophy not only gives us an insight into the clinical phenomenon as such, but also enables us to perceive its true significance.

The question naturally arises here, just as when we were considering the theory of uræmia, what substances are the chief occasion of the rise in arterial tension. This question, however, cannot at present be answered. We can merely say that the results of experiment indicate that urea probably does not play the most essential, and certainly not the sole, part in the matter. As regards the form of the cardiac hypertrophy developing in nephritis, it is primarily a so-called concentric hypertrophy, as no reason exists for an increased filling of the chambers of the heart. We therefore notice an increased force of the apex beat before we observe an increase in the cardiac dullness. Only later, when the heart muscle begins to fail, is a dilatation of its cavities added to the hypertrophy. It should furthermore be noted that while the hypertrophy always affects the left ventricle chiefly, yet, as a rule, the right ventricle, even if later, is also found to be hypertrophied. If we assume that there is a direct irritation of the myocardium (*vide supra*), this condition will not seem remarkable. On the other hand, Pässler has emphasized the fact that the right heart in nephritis then becomes hypertrophic only when the signs of muscular insufficiency of the left heart become evident.

The relation between certain diseases of the vessels and diseases of the kidneys, which must likewise be considered in connection with the question of the development of nephritic cardiac hypertrophy, will be spoken of in the chapter on Contracted Kidney.

6. FUNCTIONAL KIDNEY DIAGNOSIS

As in diseases of other organs, an attempt has been made in diseases of the kidney to elaborate a functional diagnosis, aside from an anatomical one, or the recognition of any anatomical changes in the organ. This concerns itself with a determination of the more exact nature and the degree of the disturbances in the physiological functions of the kidney. A vast amount of painstaking work has been devoted to this object, and many valuable points have thereby been brought to light. The conditions, however, are so complicated that it is difficult to deduce useful definite rules for practice. In what follows, therefore, we are only able to give a brief review of the experiments made in this direction for the purpose of a temporary orientation. In practice, we must, in general, be satisfied as heretofore with the determination of the total amount of urine excreted, and its specific weight, and with the examination of the abnormal constituents excreted.

1. **Cryoscopy.**—The freezing point and likewise the boiling point of a solution is dependent upon the number of molecules present in it. The fewer solid constituents the kidney excretes, the less will the freezing point of the urine be below the freezing point of distilled water. Under normal conditions this lowering (usually indicated by Δ) is about 1° to 2.3° C. below 0°. If it is less than 1° C., an insufficiency of the kidney is probable. Still, the findings are rarely definite, and cryoscopy, in general, especially in urine free from albumen, is not of much more value than the determination of the specific weight. If, however, the urine contains albumen, then, on account of the heavy albuminous molecules, the specific weight rises more than the molecular concentration would warrant, and cryoscopy will give a more correct idea of the excretion of solid substances than the determination of the specific gravity. Great hopes were built upon the separate examination of the urine secreted by each kidney, and obtained by means of ureter catheterization or other methods. These hopes, however, have not been entirely realized, since the two kidneys, even under normal conditions, do not functionate alike. The cryoscopy of the blood has also been considerably utilized to determine the activity of the kidneys. Under normal conditions, the molecular concentration of the blood shows only very slight variations, so that the freezing point of blood (usually indicated by δ) varies very slightly, and is generally more or less exactly 0.56° C. below that of water. In disease of the kidney, if the excretion of the solid urinary constituents is interfered with, the molecular concentration of the blood is increased, and δ sinks noticeably. In uræmia it has been found as low as 0.70° to 0.75° C. On the other hand, in nephritis and even in uræmia, normal values have also been found. This is obviously due to the fact that the body, despite the existing difficulties, always attempts to keep the molecular concentration of the blood as near normal as possible by dilution of the blood with water, by excretion into dropsical exudate, and by the retention of residual products of metabolism in the tissues. Variations of δ are therefore of more significance than normal readings.

2. **Resistance to Electric Conductivity.**—The resistance to electric conductivity of a fluid depends upon the amount of electrolytes—i. e., dissociated ions of the inorganic salts—contained therein. Increased concentration of the salts adds to the electric conductivity. In nephritic urine the conductivity is therefore often diminished. Analogous conditions apply to the blood. The results of numerous determinations are not without interest, especially in combination with the cryoscopic examinations. No important new points of view have, however, developed from this.

3. **The excretion of water and the solid urinary constituents** suffer to a great extent in the various forms of nephritis, and efforts have not been lacking to find a firm basis in this connection for the determination of the kidney function. The complexity of the existing conditions renders it difficult, however, to establish any general rules. The excretion of water is relatively easiest to observe. In many cases of acute and chronic nephritis the excretion of water is decidedly lowered, and even an increased intake of water does not increase the total quantity of urine. The water taken is only slowly excreted, or it accumulates in the tissues and increases the œdema. In other cases of kidney disease, however (*vide infra*, contracted kidney), an increased excretion of water takes place. The excretion of the nitrogenous substances in the urine

is likewise frequently, but by no means always, disturbed. In individual cases, the N excretion also frequently shows great variations. With an increased ingestion of nitrogen, the increase in the N excretion in kidney disease occurs, in general, more slowly than in the healthy organ. In severe cases there is certainly a retention of nitrogen in the body. Special mention should be made of the findings of H. Strauss relative to the increase in the residual nitrogen (i. e., the nitrogen of the blood not in albuminous combination) in cases of severe nephritis in which uræmia can readily develop.

The excretion of uric acid is often comparatively least disturbed. Of the inorganic constituents of the urine, the greatest attention has been paid to chlorid of sodium. Here, also, greatly varying conditions are found; still, as a rule, it is precisely the NaCl excretion, which is often much diminished. Since, however, the blood and the fluid of the various organs are restricted to a NaCl percentage, which fluctuates within very narrow limits ("physiological salt solution"), in continued and even increased NaCl ingestion and insufficient NaCl excretion through the kidneys, the body must find other means of elimination. This explains the relation, often emphasized, of the NaCl excretion to the development of œdema (*vide supra*). We can then make the general statement that the NaCl excretion is little disturbed in cases of nephritis without œdema, and that it is markedly disturbed in such as are accompanied by œdema. In the latter case a voluntary increase in the NaCl ingestion may therefore lead to an increase of the œdema, and a diminution in the NaCl intake to its decrease. To deduce from this that the decreased NaCl excretion is the main cause of the œdema is not correct. On the contrary, frequently the œdema formation, owing to the abnormal permeability of the vessels (*vide supra*), is certainly one of the causes of the diminished quantity of sodium chlorid in the urine.

4. **The elimination of foreign substances through the kidneys** may also be used to test the functional activity of these organs. Indigo-carmin and methylene blue have frequently been used for this purpose. After an injection of a Pravaz syringeful [about ℥xiij] of a five-per-cent solution of methylene blue, the urine, under normal circumstances, will be colored blue in about an hour. In a day and a half to two days the dye is completely eliminated. In nephritis the excretion begins later, and lasts a much longer time (several days). Similar results are observed with iodid of potassium. After the administration of 7.5 gr. (gm. 0.5) of KI, the excretion of iodin in the urine lasts about one to one day and a half in healthy persons, while in nephritics it may be extended to four or five days (F. Müller).

5. **Phloridzin Test.**—As the occurrence of glycosuria after a phloridzin injection, $\frac{1}{12}$ gr. (gm. 0.005)—i. e., a syringeful of a solution of 4 gr. (gm. 0.25) of phloridzin, 3 gr. (gm. 0.2) of sodium carbonate, and 2 ounces (gm. 50) of water—is dependent upon the function of the kidney epithelium, the action of phloridzin in nephritics has been often studied. In many cases a diminution, or even a complete absence, of phloridzin glycosuria, has been found. The test has as yet been of no great practical importance, aside from the surgical question as to the justification of a unilateral nephrectomy.

CHAPTER II

ACUTE NEPHRITIS

(Acute Bright's Disease)

Ætiology.—Acute nephritis, like most of the other forms of nephritis, is not a disease whose ætiology is uniform. The same anatomical change, which we term "nephritis," and which is attended by about the same morbid phenomena, may be excited by influences of very different kinds. Almost all these influences have one thing in common—namely, that, as we have stated in the preceding chapter, they reach the kidneys by way of the circulation and are here in part eliminated, and thus exert their specific injurious action upon the parenchyma of the kidneys; but they differ considerably from one another in their precise chemical nature. Since the pathological change in the kidneys depends upon the amount of the noxious material, upon the intensity of its action and the duration of its influence, we see that the cases of nephritis that arise in this way must present a perfectly continuous series from the mildest to the severest, from those that pass off rapidly to those that last perhaps for years and years. The history of renal pathology teaches us in the plainest way that all attempts to divide the forms of nephritis into different clinical and pathological "varieties" cannot be strictly carried out. The more scientific experience increases, the more numerous must be the forms established, and still we only too frequently have to assume all sorts of "transitional forms" merely to bring the reality into harmony with the scheme. It therefore corresponds merely to our practical needs if we take certain types from this whole list and divide nephritis into various groups; for, from the nature of the case, there can be no question of a sharp separation of the various forms.

We accordingly call those inflammatory renal affections acute nephritis which arise comparatively rapidly from any of the injurious influences soon to be enumerated, and which terminate, after a few days or a few weeks, either fatally or with recovery; or occasionally, after a rapid onset of this sort, pass gradually into a chronic form. Acute nephritis, on the one hand, follows immediately, without any fixed boundary, the mildest morbid changes in the kidney, which are usually not termed actual nephritis, but simple "parenchymatous degeneration"; while on the other hand it shows a continuous transition to those forms which last for several weeks or months, or longer, and hence are called subacute or subchronic nephritis.

Let us now consider more minutely the conditions under which acute nephritis develops. In the first place, it occurs with very great frequency as a sequel or complication of all sorts of infectious diseases. We may assert that there is really no acute infectious disease in which a secondary acute nephritis may not occasionally appear. There are, however, many diseases in which this complication is peculiarly frequent and characteristic. The exact causes of its development are as yet imperfectly known. Formerly, authorities were inclined to assume that the original germs had directly invaded the kidney itself, but probably this is true in only a few cases, if we except genuine metastatic pyonephritis. According to the views of the present day, it is much more likely that abnormal chemical substances, toxins, which develop in the body under

the influence of the infection, reach the kidneys and excite disease in them. The amount and character of these substances determine, of course, the severity of the renal disease. Since we have already dwelt upon the occurrence, the frequency, and certain peculiarities of secondary nephritis in the description of the different infectious diseases, a brief recapitulation of the facts, which have already been for the most part discussed, will suffice here.

The infectious disease which most frequently gives rise to an acute nephritis is scarlet fever. As has been shown previously, the renal affection appears but rarely at the beginning of the disease, and then in a very mild form, while the special severe scarlatinous nephritis usually attains its development only toward the end of the third week of the disease. In measles, secondary nephritis is very much rarer than in scarlet fever; in rötheln it is only of very exceptional occurrence. It is commoner again in smallpox, especially in the severe hemorrhagic forms. In varicella, renal affections are very rare, but they have been occasionally observed. They are always of but slight intensity. In typhoid fever a slight albuminuria is very common, but genuine acute nephritis is quite rare. There are some cases, however, where a nephritis appears very early, and where the other typhoid symptoms are so crowded into the background by it that we have decided difficulty in making the diagnosis of typhoid; this is called the "renal form of typhoid fever." In typhus and recurrent fevers severe cases of nephritis are not especially common, but they are seen more frequently than in typhoid fever.

The nephritis that often comes on in cholera is of great practical importance. This is seen in the earlier stages, and is especially one of the most frequent causes of the so-called cholera typhoid (see page 103). Of course it may appear questionable whether the renal affection here is always of a genuine inflammatory nature, or develops only in consequence of the disturbance of circulation.

Nephritis develops quite frequently in the course of diphtheria, especially in severe cases of this disease; but the renal affection only rarely reaches a high degree. We sometimes see, however, very severe forms of nephritis in all the so-called septic diseases (septic nephritis, see page 138), in acute ulcerative endocarditis and endocarditis verrucosa, and allied affections, such as puerperal fever, septic wounds, etc. Erysipelas also is very often associated with nephritis, and likewise croupous pneumonia; while, on the other hand, acute articular rheumatism is seldom accompanied by albuminuria. It is not very exceptional for sore throat, particularly follicular tonsillitis, to occasion acute nephritis. It is of great practical importance to know that a severe secondary nephritis may follow primary diseases of this sort, even if the latter seem to be very mild. In every seemingly primary acute nephritis we should therefore look for a possible previous tonsillitis. Following tonsillitis, acute infectious inflammations of the nasopharynx may occur, particularly in children. These, as I have repeatedly observed, sometimes lead to severe acute hemorrhagic nephritis, usually associated with pronounced acute swelling of the lymph-glands of the neck. Much more rarely than the diseases of the pharynx do the acute infectious and toxic gastric and intestinal diseases lead to a secondary nephritis.

We ought also to speak of the acute nephritis sometimes seen in patients with pustular eruptions, such as impetigo, pustular eczema, severe scabies, etc. Probably we have to do in most of these cases with a mild septic infection, which has found ingress through one of the many minute scratches or other lesions of

the skin. We must also bear in mind the possibility (*vide infra*) of damage done by external applications which are absorbed by the skin.

There are many chronic infectious diseases besides these acute ones, in the course of which acute nephritis may appear. We see this complication most frequently in the case of primary tuberculosis, but it seems to us highly probable that the nephritis in this instance is not directly connected with the tuberculosis, but is the result of absorption of septic matters from the pulmonary cavities and similar places. Syphilis should also be mentioned in this connection. We have ourselves repeatedly seen mild and even severe acute nephritis develop in the secondary stage of syphilis. Finally, malaria might be mentioned, although the nephritis which occurs in this connection usually takes a chronic form.

Besides the infectious forms of nephritis just described, there is a second great group, which may be classed under the general heading of toxic nephritis. In these cases we are dealing with the deleterious action of definite chemical substances which enter the body from without and are excreted from it by the kidneys. It is wholly impossible to enumerate all the substances which have this injurious effect; we will therefore confine ourselves to mentioning those of the greatest practical importance. Among the poisons proper we may mention the mineral acids, sulphuric, hydrochloric, and nitric acids, oxalic acid, phosphorus, arsenic, lead, mercury (corrosive sublimate), and chromate of potassium. Among remedies used internally, which may excite nephritis when given in too great doses, we may mention cantharides, squills, balsam of copaiba, turpentine, salicylic acid, and chlorate of potassium. This last causes hemoglobinuria as well as nephritis. It is also very important to know that many remedies applied externally are absorbed by the skin, and that in this way they may reach the kidneys and excite severe changes there. Among these are cantharidal plaster, preparations of tar, petroleum, styrax, naphthol, and pyrogallic acid. We would like to call attention here to what, in our opinion, is no insignificant danger—namely, the effect of too active and long-continued mercurial treatment upon the kidneys. We must mention, in addition to these, the nephritis which may arise from the too abundant use of carbolic acid or iodoform on the surface of open wounds. This has been repeatedly observed, and it might be stated as a general fact, that acute nephritis is by no means uncommon as a result of the careless use of drugs, such as arsenic, bichlorid of mercury, carbolic acid, etc. At any rate, every conscientious physician must keep this danger constantly in mind! Under some circumstances renal affections may even arise in individual cases from taking excessive amounts of certain foods and drinks, such as spices, alcohol, horse radish, or very acid foods.

Aside from the forms of acute nephritis thus far discussed, there is also a type where the acute nephritis appears as an apparently primary disease in persons previously healthy. In many such cases we are absolutely unable to make out any causative factor, while in others, again, we do find certain unfavorable influences affecting the patient, such as hard drinking or severe labor in the open air. It cannot be doubted that exposure to severe cold and thorough wetting promote the development of acute nephritis. The author's experience has convinced him of this fact. Thus, the disease may occur when a man works in the snow or in cold water. We possess as yet, however, scarcely any knowledge of the conditions which bring about this result. Many investigators

assume that cold interferes with the excretory function of the skin; others imagine that the blood is damaged as it circulates in the cutaneous vessels. We have repeatedly seen cases of apparently primary acute nephritis in great beer drinkers. We suppose that in such cases the effect of the long-continued chronic intoxication with alcohol is cumulative, and finally leads to the apparently spontaneous acute development of nephritis (acute alcoholic nephritis). Sometimes, however, even in such cases there seems to be some other factor, such as catching cold, or an infection which gives the final impulse to the development of the disease.

A considerable number of cases of apparently primary acute nephritis must, however, be regarded in the last analysis as the results of a septic infection of some sort. If we question the patient closely, we learn in such cases that the appearance of the nephritic phenomena has often been preceded by symptoms of a mild sore throat which scarcely attracted the patient's attention, or an insignificant gastrointestinal disturbance, etc. Such statements are indicative of the possible place of infection. Again, slight purulent affections of the skin, such as furunculosis, phlegmon of a finger or toe, and eczema may sometimes furnish a gate of entrance for infection. In considering all cases of apparently primary idiopathic and acute nephritis, it is very important to remember that not infrequently a chronic nephritis may exist for a long while, perhaps without any symptoms whatever, and suddenly flame up into an acute disease (acute recurrent nephritis of Wagner), and thus simulate a primary acute nephritis. We cannot get at the correct interpretation of such cases without careful inquiry into the previous history, and sometimes only after observing the subsequent course of the disease.

In conclusion, we have an especial form of acute renal disease to mention in this connection—viz., the nephritis of pregnancy (*nephritis gravidarum*). This does not usually appear until the last months of pregnancy. It may attack women whose health was previously perfect, and it is much more frequent in primiparæ than in multiparæ. The precise causes of the nephritis of pregnancy are very obscure. The earlier explanations, according to which the renal disturbance was occasioned by pressure of the pregnant uterus upon the renal arteries, the renal veins, or the ureters, are entirely unsatisfactory. It is probably due to the action of toxins, the formation of which is dependent upon specific processes of pregnancy, such as placental metabolism.

Pathological Anatomy.—The anatomical changes of acute nephritis show a continuous series from the mildest to the severest degrees, according to the intensity of the injurious action. The mildest changes, which, as we have said, are not called actual "inflammation," but usually simple parenchymatous degeneration, affect exclusively the parenchyma of the kidney—that is, the epithelium—while the interstitial tissue, the connective tissue, and the vessels remain perfectly normal. This fact is of prime importance, since it implies that, in almost all the injurious influences acting on the kidneys, the specific renal parenchyma itself is diseased first and before any other. On macroscopic examination, the kidneys of "parenchymatous degeneration" may show scarcely any plainly perceptible changes, but it sometimes strikes the practiced eye that the kidneys are a little enlarged, that the cortex on section shows either a more reddish-gray, dimmed coloring (cloudy swelling), or a more grayish-white, yellowish hue (fatty degeneration). The microscopic examination gives

more accurate information as to the degree and the extent of the disease. We distinguish different conditions according to the form of change in the epithelium, of which the three following are most important: 1. *Cloudy swelling:* It is most easily made out in the epithelium of the cortical tubules, but it may also be seen in the epithelium of the glomeruli. The cells swell, their contents become uniformly granular and cloudy, the nuclei swell, and finally disappear. Such changes are often found in acute infectious disease, such as typhoid, smallpox, and diphtheria. 2. *Fatty degeneration:* This may proceed from the cloudy swelling, or may develop independently. Many fat drops appear both in the cells of the uriniferous tubules and also in the epithelium of the glomeruli, and they may finally lead to the disintegration of the cells. Simple fatty degeneration of the kidneys is sometimes found in acute infectious diseases, after certain poisons, such as phosphorus, and finally in anæmic conditions. 3. *Necrosis of the Renal Epithelium:* The nuclei of the cells disappear, and the cells are changed to clear homogeneous flakes, while in some cases they are greatly swollen ("dropsical degeneration" of Nauwerck-Ziegler). Genuine epithelial necrosis is found in the kidneys, chiefly after the action of toxic substances—cantharides, the chromic and chloric salts, etc.—but sometimes also in infectious diseases. Combinations of simple necrosis with granular cloudiness and fatty degeneration are not infrequent. Both the last-named states may undergo resolution if they have not reached a high degree. Otherwise all the degenerations mentioned lead to the destruction and disintegration of the cells; nevertheless, a complete restoration is possible, from the regeneration of new epithelial cells from epithelium that is still present.

We term those changes in the kidneys genuine acute nephritis, in which not only the renal parenchyma proper, the epithelium, but also the interstitial tissue, especially the vessels, is affected; so that we can make out the exudative changes characteristic of all inflammatory processes—the escape of fluid and cells from the vessels. In these cases the different histological processes may be combined in the most varied ways, so that the anatomical picture presents quite great variations, although it is almost always seen on analysis to display the same processes.

If we begin with the histological lesions in acute nephritis, in order to learn to recognize at once the essential changes, we have first precisely the same processes of degeneration in the epithelium which have been already described, but they are usually present here in a more marked degree. In some cases the simple necrotic processes predominate; in others, the fatty degeneration. We often find degenerated cells, and not infrequently a more or less marked desquamation of epithelium. We see also the special inflammatory changes. We find a fluid inflammatory exudation, rich in fibrin, and therefore soon coagulating in the interstitial connective tissue, which is dilated and swollen by it—inflammatory œdema. The same exudation is also found in the uriniferous tubules, and, by the proper methods, by alcohol or by boiling the fresh kidney, the albuminous effusion can be made out both in the capsules of the glomeruli and in the uriniferous tubules. The interpretation of the exudation is, of course, made very difficult, or often wholly impossible, by the presence of albuminous urine in the uriniferous tubules. The second characteristic of inflammation, the "cellular exudation"—that is, the emigration of white blood corpuscles—is also present. In the interstitial tissue we find accumulations of round cells, usually

distributed in foci, and white blood corpuscles in greater or less numbers also enter the interior of the uriniferous tubules. We often find many hyaline casts in the lumen of the straight tubules or of Henle's loops, whose origin is, in all probability, connected with the albuminous exudation and the emigrated white blood corpuscles (see page 729). The vessels themselves are often hyperæmic and dilated, but in some cases they are compressed by the interstitial inflammatory œdema. It is of special significance that in very many cases there are hemorrhages, either into the interstitial tissue or into the interior of the uriniferous tubules, or even into Malpighi's capsules.

Special care has been recently devoted to the study of the minute changes in the glomeruli, although at present there is a conflict in the views regarding them. Klebs called attention to the fact that in scarlatinal nephritis the lesions may be almost exclusively confined to the glomeruli (glomerulo-nephritis). Sometimes kidneys of this sort seem almost normal to the naked eye, although during the life of the patient there have been the severest symptoms, such as anuria and uræmia. Friedländer, Ribbert, Langhans, and others have pursued the study of the histological changes in the glomeruli. In part there are degeneration, hyperplasia, and desquamation of the glomerular epithelium; in part changes in the walls of the blood vessels themselves. With regard to the secretion of urine, and, in particular, the development of albuminuria, these changes in the glomeruli are certainly of the greatest importance, and it is not improbable that in many cases of acute nephritis the disease begins chiefly in the glomeruli; but, on the other hand, it is not proper to suppress too completely the other changes in the renal parenchyma as compared with the glomeruli-nephritis.

In regard to the extent of all these changes which have been described, it should be noted that not infrequently certain portions of the kidney are more severely diseased than others, so that sometimes the nephritis tends to develop in separate foci. In general, however, acute nephritis is diffuse.

If the histological processes have been made clear, the understanding of the microscopic appearance of the inflamed kidney is very simple. We can understand that either this or that "form" of acute nephritis must be present according to the predominance of this or that histological process. If an abundant interstitial exudation is present, the kidney is much enlarged; if this exudation is slight, the kidney varies but little, or not at all, from its normal size, notwithstanding any other severe changes. In the first case it usually feels soft, from inflammatory œdema; in the second case, it is comparatively firm. If there is a marked hyperæmia of the kidney, it appears much reddened; if the kidney is anæmic, it is paler; and if an extensive fatty degeneration is also present, it is yellowish white or yellow. If hemorrhages are present, they can easily be recognized with the naked eye on the outer surface beneath the capsule as dark red points that cannot be wiped away. We speak then of an "acute hemorrhagic nephritis." On section, the medullary substance is more or less swollen, often projecting somewhat above the general level of the cut surface, its normal striated appearance is almost always obliterated, and its color shows the same variations as the outer surface of the kidney. Not infrequently the diseased Malpighian corpuscles can be recognized by the naked eye as grayish-red or whitish points. Since, as we have said, the nephritic changes often show not a uniform and diffuse, but a

nodular arrangement, we can understand that the kidneys sometimes have quite a mottled appearance, since hyperæmic or hemorrhagic red spots alternate with the lighter anæmic and the yellow fatty-degenerated parts.

There are, accordingly, cases of nephritis which show almost nothing abnormal to the naked eye, while, on the other hand, there are hemorrhagic and nonhemorrhagic forms, appearing pale yellow, red, or variegated, none of which can in the essential features be separated from one another, but which are combined with one another in all conceivable ways. The forms of nephritis that differ in ætiology have, to a certain degree, definite and characteristic anatomical types, but strict rules cannot be laid down in regard to this.

Clinical History.—The most essential symptom of acute nephritis is the abnormal character of the urine. In most of the milder, and even in many of the severer cases of nephritis, the change in the urine is the sole objective clinical symptom which renders the diagnosis possible. The physician must, therefore, make it his practice to submit the urine to repeated examinations in every case of disease where there is any possibility of the presence of a nephritis.

The simple parenchymatous degeneration of the kidneys, cloudy swelling, fatty degeneration, etc., may probably sometimes exist without being followed by any discoverable change in the urine; but they often lead to a slight albuminuria, dependent upon the change in the epithelium of the glomeruli. If, then, the urine contains a slight amount of albumen (which usually soon passes off) in the course of any febrile infectious disease or other affection (the so-called febrile albuminuria, etc.), we are justified in assuming some of these mild conditions of degeneration in the kidneys. Sometimes we find in the sediment of the urine a few hyaline casts, a few white blood corpuscles, etc. As we have repeatedly stated, these conditions pass into nephritis proper without any sharp limitations.

Character of the Urine in Acute Nephritis.—In almost every case of severe nephritis the amount of urine for the twenty-four hours is more or less diminished. This is caused directly either by the lessened elimination of water by the kidneys, or by the plugging of many uriniferous tubules by casts, desquamated epithelium, etc. The amount evacuated daily is often only 15 or 20 ounces (400 to 700 c.c.), but it sometimes falls to a much lower figure, 2 or 3 ounces (100 to 50 c.c.), and there may finally be even complete anuria. In general, though not without exceptions, the diminution of the amount of urine runs parallel to the severity of the anatomical changes in the kidney. Improvement in the disease is very often first seen in an increase of the amount of urine. If there was a previous œdema, and this is absorbed, the daily amount of urine often rises during convalescence to a very considerable quantity, 80 to 100 ounces (2,500 to 3,000 c.c.). Even in cases in which there had been no decided œdema, we have often seen during recovery from acute nephritis very abundant diuresis. In so far as this symptom did not relate to the excretion of water that had been retained in the system, it might perhaps be referable to an abnormal permeability of the glomeruli, similar to the condition sometimes seen after a severe attack of typhoid fever.

The specific gravity of the urine is at first usually increased, since the urine is poor in water, but comparatively rich in solid constituents, especially in albumen (*vide infra*). Of course there are great differences here, and a

urine secreted in an abnormally small amount may show a specific gravity of only 1.010 or 1.015, while, on the other hand, urines with a specific gravity of 1.020 to 1.030, or even more, have been observed. If during convalescence a very abundant, watery urine is passed, it of course usually has a low specific gravity, about 1.005 to 1.008.

In many cases, but of course not in all, we may suspect the abnormal character of the urine from its appearance. This depends chiefly upon an admixture of abnormal morphological constituents. If these are present in large numbers, as is usually the case, the freshly passed urine is cloudy, and deposits a more or less abundant floccular sediment. The appearance of the urine is most altered if blood be mixed with it (hemorrhagic urine). According to the amount of blood, the urine is a light or dark red, or even a dark black-red, and often has a greenish reflection when the light falls on it.

The microscopic examination of the sediment gives more accurate information upon the different morphological constituents. In general, it may be said that in most of the severe cases of acute nephritis the urine contains numerous casts of the hyaline and other varieties. As has been already mentioned, the epithelial casts and the firm, waxy casts are especially common in the severer forms of acute nephritis. The hyaline casts have usually adherent to them red and white blood corpuscles, as well as epithelium, fat drops, and detritus. These morphological constituents are also found free in greater or less amount. The sediment is apt to contain also numerous beautiful crystals of uric acid.

Individual cases are often characterized by a striking predominance of some one constituent—epithelium, white blood corpuscles, or red blood corpuscles—but no special rules in regard to this can be given. We have spoken previously (page 731) of the special conclusions we can draw from the different objects found in the sediment. We can accordingly distinguish an acute hemorrhagic, or nonhemorrhagic, an acute desquamative, and a fatty degenerative nephritis, but we must always bear in mind that all these forms pass into one another without sharp boundaries.

The chemical examination of the urine gives, as the most important and constant result, usually a considerable amount of albumen. Since the reaction of the urine is almost invariably acid, the albumen is immediately precipitated on heating the urine, and sinks to the bottom of the test-tube, where it usually takes up about one half or three fourths of the volume of urine used for the heat test. More accurate quantitative determinations of albumen give most frequently in acute nephritis an amount of albumen of from three tenths to one per cent; higher percentages are rare. The daily total amount of albumen eliminated amounts to about 1 or 2 drachms (gm. 5 to 8), or sometimes more, but the daily loss of albumen from the body hardly ever exceeds the amount of 5 drachms (gm. 20). The variation in the amount of albumen eliminated in different cases is quite noticeable.

The examination of the other solid constituents, which is not generally employed in practice, usually gives a diminished secretion of urea, sodium chlorid, phosphoric acid, etc., corresponding to the diminution in the whole amount of urine (*vide supra,* page 743).

The Other Symptoms of Acute Nephritis.—Local symptoms in the kidneys appear only exceptionally to any extent. There is, of course, a cer-

tain tenderness in the region of the kidneys, which, however, is too ambiguous to have very great symptomatic importance. It is more often the case that the abnormally concentrated urine causes frequent micturition, associated with a disagreeable burning sensation—a sort of vesical tenesmus.

The subsequent symptoms of acute nephritis, which appear in the rest of the body, and among which dropsy takes the first place, are far more important than the local symptoms. Œdema may be entirely wanting in acute nephritis, particularly in cases of secondary nephritis after pneumonia, erysipelas, diphtheria, and usually in cases of septic nephritis. On the other hand, œdema is especially characteristic of many cases of acute nephritis, including cases due to scarlet fever, to catching cold, to alcoholic excess, and to pregnancy, as well as the so-called primary nephritis and the nephritis which follows cutaneous diseases, such as scabies or pustulous eczema. This œdema is not infrequently the most prominent clinical symptom. We must always be prepared for its appearance, especially when the amount of urine shows a considerable and persistent diminution.

The œdema is usually discovered first in the face, which has a bloated and often a pale and somewhat shiny appearance. The eyelids are usually most swollen at first. Besides the face, the ankles, the legs, the scrotum, and the dependent parts of the trunk may be the chief seat of the œdema, the severity and extent of which may, of course, vary greatly in different cases. If a high degree of general dropsy develops, this is a source of great distress to the patient. The movements of the body are much restrained, and all changes of position are difficult, associated with great exertion, and painful. In the severest degrees of dropsy small fissures may form here and there in the excessively tense skin, from which the dropsical fluid oozes. Such little wounds are sometimes the starting point for disagreeable erysipelatous inflammations, etc.

If great œdema of the skin is present, we usually find at the same time a more or less marked dropsy of the serous cavities. It is often hard, however, to make out ascites or hydrothorax on physical examination, owing to the œdema of the skin that is present. The symptoms mentioned acquire their chief clinical significance from the difficulty of respiration necessarily associated with them, since the diaphragm is pressed upward by ascites, and the lungs are compressed by hydrothorax. If a hydrothorax is more marked on the left, or especially if hydropericardium sets in, the activity of the heart is materially impaired.

A marked œdema of the mucous membranes develops but rarely; in a few cases we have seen œdema of the conjunctivæ, œdema of the soft palate, and œdema of the glottis. Of the œdemas of internal organs, œdema of the brain has already been mentioned as a possible cause of severe nervous uræmic symptoms. Œdema of the lungs, which often comes on toward the end of the disease, when it terminates unfavorably, is usually not to be regarded as a part of the general œdema, but as a result of the final cardiac weakness.

In regard to the other symptoms in the different organs, the symptoms on the part of the circulatory apparatus must first be mentioned. The pulse is often abnormally tense, hard, and full (see page 741). In the beginning of the disease it is often somewhat slow; later it is usually accelerated. A beginning cardiac hypertrophy can often be made out postmortem, and some-

times clinically, in cases which have lasted a rather long time—four to six weeks. It seems to develop most rapidly in children who were previously well and strong. We pay especial regard to the character of the apex beat and to the accentuation of the aortic second sound. The occasional nosebleeds are probably connected with the increased arterial tension. Pericarditis is seen as a rare complication—a complication which is connected with the general fact that in all forms of nephritis the different internal organs, especially the serous membranes, have a tendency to inflammation.

Of the symptoms in the respiratory apparatus, we have mentioned above the dyspnœa consequent upon the dropsical symptoms. In severe cases the lungs themselves are often drawn into sympathy, as shown by the development of a diffuse bronchitis or a peculiar form of pneumonia. The latter stands midway between a catarrhal and a croupous inflammation. It exhibits, to a certain degree, a form of stiff inflammatory œdema, and occurs in just the same way in the chronic forms of nephritis as in acute nephritis. When it involves both lungs to a great extent, it may be the immediate cause of death. The development of general pulmonary œdema is usually a sign of beginning weakness of the left ventricle, as we have said above, although here, too, inflammatory factors may have some influence.

Vomiting is the most important symptom in the digestive apparatus. If it appears in a marked degree, it may almost always be considered as a uræmic symptom, and then is often the precursor of severe nervous symptoms. The appetite is almost always diminished in acute nephritis; the bowels are usually constipated, but there may be quite severe diarrhea. We may mention peritonitis, which is sometimes purulent, as a very rare complication (*vide supra*).

The temperature is markedly influenced by acute nephritis only in those cases where the disease develops in previously healthy persons, or at least in those free from fever. Then we see quite frequently a moderate fever, with an irregular rise of temperature to about 100° to 102° F. (38° to 39° C.). It is quite rare that an apparently primary acute nephritis begins suddenly with a chill and high fever, 104° F. (40° C.). The behavior of the temperature on the onset of uræmic symptoms has already been described (page 739).

The state of the general nutrition suffers in quite a noticeable degree in most of the severe cases of acute nephritis. The emaciation is often concealed by the œdema; but the anæmia is the more prominent, and often lends to the bloated face a peculiar aspect.

Uræmic symptoms may come on at any time in the course of acute nephritis. We are often prepared for the onset of uræmia by a previous marked decrease in the secretion of urine, or by the well-known prodromal symptoms, but in other cases it begins very suddenly with severe symptoms of eclampsia. In regard to all further details we may refer to what was said on page 734 *et seq.*

The Course and Different Forms of Acute Nephritis.—The whole clinical picture of acute nephritis depends very materially upon the form of its development. If an acute nephritis comes on in the course of a severe infectious general disease, as in the course of a septic affection, of ulcerative endocarditis, or of severe typhoid, the changes in the urine are often the sole indication of the occurrence of the complication. The type of the severe

febrile general disease is in no way materially modified by the added renal affection; œdema and uræmic symptoms do not usually appear, often because the primary disease soon ends in death.

Also when nephritis comes on in previously healthy persons or in chronic invalids, the tuberculous, etc., in many cases the changes in the urine are the chief symptom, while the other general and secondary symptoms are scarcely evident at all, or at least only in a very slight degree. Such mild cases are associated only with more or less general dullness and loss of appetite. Œdema is entirely absent, or present only to a very slight degree. Of course such cases demand great caution, since even in them we may have a sudden outbreak of severe uræmic symptoms.

The fully developed type of severe acute nephritis is seen especially in scarlatinal nephritis (*q. v.*); it is also seen in many cases of apparently idiopathic nephritis (*vide supra*), or nephritis coming on after exposure to cold, etc. In these cases there is often the development of a general dropsy, secondary pulmonary affections, uræmic symptoms, the symptoms mentioned in the circulatory apparatus, etc. In these cases, too, the examination of the urine affords the only certain means of judging accurately of the condition, but the other morbid symptoms which appear early—œdema, anæmia, and vomiting—may direct our suspicions to the developing renal affection.

Scarcely any general statements can be made as to the course and the duration of acute nephritis, since the variations in this respect are too great. We will refer to the description of the different primary diseases, in which the characteristic marks of any renal complication are always stated. A few remarks must be added about some of the simple forms of acute nephritis.

The so-called *primary idiopathic nephritis* usually appears rather suddenly without any demonstrable cause, or it may follow a marked chill ("nephritis due to catching cold"). The first symptoms of the disease are sometimes insignificant, but at other times they are quite severe—chills, fever, renal pain, etc. Sometimes other "rheumatic symptoms," such as angina or articular pains, are also present. Often the first thing which attracts the attention of the patient is the occurrence of œdema, the puffiness of the face, or slight shortness of breath due to hydrothorax. Sometimes the first symptom is vomiting. The further course of the illness may be mild or severe. In the former case the œdema that has appeared is but slight, the changes in the urine (albuminuria, hematuria, etc.), do not attain a very high degree, and after a few weeks complete recovery ensues. In other cases, however, the type of a severe, acute, and very often hemorrhagic nephritis develops, with great general dropsy, uræmia, etc., which in three or four weeks, or sooner, may lead to death; but improvement may follow in spite of the severest symptoms. Then the amount of urine gradually increases, and the abnormal constituents of the urine, the œdema, and the other morbid symptoms, gradually disappear. Of course, it is often a long time before complete recovery ensues, since, even when the patient feels perfectly well subjectively, the urine may still sometimes contain some albumen, a few casts, or a few red blood corpuscles. In such cases one must think of the possibility of a transition of acute into chronic nephritis. Not infrequently there is apparently complete recovery, but several weeks or months later there appears suddenly, perhaps after some special cause, such as catching cold, or overexertion, or some febrile dis-

ease, a fresh attack of acute nephritis ("acute recurrent nephritis," *vide supra*).

The *nephritis of pregnancy* usually begins gradually. Frequent micturition and œdema of the lower extremities make their appearance, and besides these there are often nausea and even vomiting. Frequently these symptoms are so slight that the changes in the kidneys are easily overlooked. If we examine the urine, however, we usually find it quite rich in albumen, but comparatively poor in morphological elements. The slight sediment consists of hyaline casts, a few white blood corpuscles, and some epithelium. Only rarely does the urine assume a hemorrhagic character. If such is the case, we find in the sediment red blood cells in more or less profusion, and sometimes a considerable amount of hematoidin crystals also. Albuminuric retinitis, with the corresponding disturbances of vision, is not infrequently observed.

The condition described almost always lasts to the end of pregnancy. In the cases that proceed favorably a very rapid recovery often follows after the birth of the child; but the onset of *eclampsia gravidarum* is to be dreaded as a not infrequent and dangerous complication. It begins usually shortly before or during labor, and, very rarely, only a short time after delivery. After mild prodromal symptoms, or even quite suddenly, violent general convulsions develop, during which the child is usually born. A more or less persistent coma follows the convulsions. The convulsions may be very frequently repeated. Death ensues in about one third of the cases; the other cases usually recover, only rarely passing into chronic nephritis. The prognosis is still more unfavorable for the child than for the mother, inasmuch as the child dies in nearly one half of the cases.

The causes of eclampsia have not been entirely cleared up. Some authors look upon it as a real uræmia. Against this view is the fact that sometimes eclampsia develops without a previously existing albuminuria. It may, however, in itself lead to a mild albuminuria. It is probable that specific toxic influences play a causative rôle in eclampsia.

The prognosis of the nephritis of pregnancy, aside from eclampsia, is in general favorable. Only rarely does a mild albuminuria persist for any length of time after confinement. Then we must think of the possibility of the development of a chronic nephritis. The possibility of recurrences in the event of subsequent pregnancy must be borne in mind. The treatment should be according to the general principles in vogue for nephritis (*vide infra*). The artificial interruption of pregnancy is indicated only in the severer cases. More definite information may be found in the text-books on obstetrics.

The anatomical changes in the nephritis of pregnancy are hardly ever very striking to the eye. The kidneys are usually pale and but little enlarged. Under the microscope we usually find a slight interstitial œdema and degenerative changes in the epithelium. Only rarely are more marked nephritic appearances present.

Acute alcoholic nephritis is a term which we apply to that form of the disease which we have repeatedly observed in excessive beer drinkers—for instance, in brewers. The appearance of the disease seems to be favored by coincident exciting causes, such as catching cold, but beyond this it is to be regarded as the sudden development of a renal disease resulting from the toxic influences of alcohol habitually ingested. There is a rapid and great develop-

ment of general dropsy. The amount of urine is moderately diminished. The urine contains much albumen, but usually remains rather clear and contains no blood. As a rule, the patients are obese, and their discomfort is great. A favorable termination is possible under suitable treatment, but yet there is always danger of a transition into chronic nephritis.

Diagnosis.—Acute nephritis can be overlooked only when the examination of the urine is neglected or when it is impracticable. The latter sometimes happens, for example, when the patient does not come under observation until after the onset of severe uræmic symptoms. Otherwise, however, the changes in the urine always furnish evidence enough to recognize the existence of the affection of the kidneys. We can, of course, decide that the nephritis is acute only by consideration of the history, the ætiological conditions, and the whole course of the disease. We must also bear in mind the possibility that there may be an acute exacerbation in a chronic nephritis that has already existed for a long time, and has been perhaps without symptoms—acute recurrent nephritis, usually hemorrhagic.

Prognosis.—The prognosis of acute nephritis depends in many cases not only upon the renal affection, but also upon the underlying primary disease. Many cases of primary nephritis from toxic action, or exposure to cold, and also many cases of secondary nephritis after scarlet fever, in pneumonia, typhoid fever, or syphilis, during pregnancy, etc., recover perfectly in a short time or after several weeks, according to the severity of the individual case. On the other hand, however, it must be said that every case of nephritis must be judged with great caution, partly because it may be the starting-point of a subsequent chronic renal disease, and partly because dangerous sequelæ may sometimes develop in cases that at first are apparently mild. The dangers of acute nephritis are chiefly: First, the appearance of severe general dropsy, especially in the internal cavities of the body. Of the forms of dropsy hydrothorax is the most dangerous, as it may produce suffocation by compression of the lungs. Second, uræmia, especially in its severe convulsive forms, with high temperature and, finally, cardiac paralysis. Third, the inflammation of internal organs, among which secondary pneumonia, in particular, is a frequent cause of death, while secondary pericarditis and peritonitis, as we have said, are seen in but very few cases. We must bear in mind, however, that in individuals otherwise healthy the severe sequelæ just mentioned may also be recovered from. The most extreme dropsy may be reabsorbed, and we sometimes see recovery, especially in children, after the worst uræmic symptoms.

Treatment.—With regard to prophylaxis, the reader should be reminded that in all diseases which experience has shown to be prone to lead to secondary nephritis—for instance, scarlet fever—the kidneys should be guarded as much as possible, from the start, by suitable diet (milk); by promoting the secretion of the kidneys ("flushing the kidneys"), by administering an abundance of liquids, such as mineral waters, milk, and lemonade; and by stimulating the cardiac activity by warm clothing, warm baths, and rubbing with alcohol.

If nephritis has already developed we can scarcely hope to influence directly the inflammatory process. The remedies recommended for this purpose are tannin, uva ursi, fuchsin, and methylene blue, but to an unprejudiced observer they seem absolutely useless. Strontium lactate, in doses of 4 gr. (gm. 0.25) three times a day, in powder or solution, might be worthy of mention. Several

reliable observers claim to have seen a good effect upon the albuminuria from its use. We expect as little result at present from "external antiphlogistics" as from the internal remedies mentioned—that is, from local bloodletting, applications of ice to the region of the kidneys, etc. Only in the rare cases where severe pain in the region of the kidneys comes on at the beginning of nephritis, in an otherwise robust individual, are we at present justified in trying leeches or a few dry cups. The warm baths, to be described more fully below, have perhaps an indirect favorable action on the process in the kidneys, since they produce a hyperæmia of the skin, and thus lessen the flow of blood to the kidneys.

Although we must accordingly admit that there is scarcely any remedy at our service which has a direct therapeutic influence upon the diseased kidneys, the treatment of nephritis may nevertheless produce very significant results, since both a number of hygienic measures and the fulfillment of certain symptomatic indications are of the greatest importance.

Among the general hygienic measures we must mention first strict confinement to bed. In the severe cases its necessity is self-evident; but, even in the milder cases, which run their course without any severe subjective symptoms, constant rest in bed is necessary throughout. In this way we not only avoid the unfavorable action of cold upon the external skin, but we also stimulate the activity of the skin, which must act vicariously for the kidneys by the uniform warmth of the bed; while any useless muscular exertion, which would tax the heart's capacity for work, is also avoided by staying in bed. In general it is advisable to cover the patient quite warmly, so as to keep him in a constant slight perspiration.

The regulation of the diet is very important. All those foods and drinks which may irritate the kidneys are to be strictly avoided, especially spices, very sour substances, strong tea and coffee, or alcoholic drinks. Milk has for a long time proved itself to be by far the most suitable and best food. It has won for itself the reputation of a remedy in renal disease, and the best results have often been seen from a methodical "milk cure"—that is, from feeding the patient almost exclusively with milk. The great aversion of some patients toward milk, however, is sometimes an obstacle to its use. We may often be aided, then, by making the milk more acceptable to the patient by the addition of a little coffee, cocoa, a little cognac, or soda water. Among other foods to be recommended are buttermilk, milk gruel with rice or groats, and flour gruel. We should be very cautious about giving meat as long as there are severe symptoms. Less harmful are light broths made from pigeons, fowls, or veal. We should also, in general, restrict the amount of eggs. On theoretical grounds a restriction of the amount of salt ingested is to be recommended (*vide supra,* page 744). As a matter of fact, a salt-poor diet is preferable, especially in cases of beginning or more pronounced œdema. Perhaps the frequently tested value of milk depends in part on its slight salt content. As regards the administration of liquids, we believe their abundant, but not excessive, ingestion to be advantageous in every case of acute nephritis, and, despite all theoretical objections, even when dropsy is present. For beverages, besides milk we may give Seltzer, Wildinger, or Fachinger water, tea, lemonade, and other watery fruit drinks. Alcoholic beverages must be entirely withheld, or, at the most, we may allow a little claret. The stronger

wines are given only when the heart becomes weak, and even then their usefulness is dubious.

The chief object in the symptomatic treatment consists in preventing the injurious results of the defective elimination of the water and the solid constituents of the urine, or in remedying these results if they have occurred. This purpose can be attained only by exciting, as far as possible, the activity of other organs which in this respect may act vicariously for the kidneys. The skin deserves the first attention here, through which, by means of the sweat-glands, large amounts of water and also, to a certain extent, the solid constituents of the urine, which have been retained, may be eliminated. The diaphoretic treatment of renal diseases has, therefore, been generally in vogue for a long time. If the patient's general condition permits, we always begin with it as early as possible, even before there have been any signs of œdema, uræmic symptoms, etc. To produce diaphoresis, hot baths are especially useful and also hot packs. The former are given at from 95° to 100° F. (35° to 38° C.), or even hotter. The patient stays about a quarter or half an hour in the bath, is then rapidly dried somewhat, wrapped up in bed in a previously warmed sheet, and well covered up to the neck with blankets. In order to make the procedure somewhat easier for the patient, it is a good plan to cover the forehead with a cold compress, always to wipe the sweat carefully from the face, and frequently to give a little swallow of fresh cold water. The production of sweat is even better excited during the pack if the patient takes some hot drink, hot milk with soda water or hot elder tea. It sometimes seems to aid diaphoresis if an internal diaphoretic be given at the same time, the best being 5 to 10 gr. of Dover's powder (gm. 0.3 to 0.5), or a teaspoonful or two (5 to 10 c.c.) of liquor ammonii acetatis [U. S. P.] (*spiritus Mindereri*) in a cup of elder tea. We have found a good rubbing of the whole body with dilute warm French brandy of service before the pack. The pack may last two or three hours. Of late years, particularly in severe cases with marked dropsy, we have almost entirely abandoned baths, and in their stead employed with the best results the hot, wet pack, or dry heat (*vide infra*). For the pack, the patient is completely wrapped up in a hot, wet sheet; on both sides are arranged several jugs filled with hot water, and likewise enveloped in moist cloths; then the patient is covered with several dry blankets and is given two or three cups of some hot drink. The duration of a pack of this sort is about two or three hours. Dry heat may be applied either by special appliances (sweat boxes, "patent sweat beds"), or by the simple introduction of heated air under the bedclothes. This latter method is the most convenient and reliable. In our clinic we formerly almost exclusively used a simple tin pipe bent at an angle and fastened upon a board, with its upper end introduced under the bedclothes, which should be, if possible, somewhat raised up, while under its lower open end a lighted alcohol lamp is placed upon the floor; the warm air rises and is conducted beneath the bedclothes, where the heat becomes so great that the upper end of the pipe must always be wrapped in a cloth wet in cold water (see Fig. 98). At present, on account of its convenience and safety, the incandescent electric-light bath, which acts as a most excellent diaphoretic, has superseded all other appliances. Where it can be used, we consider it as one of the most useful adjuvants in the treatment of all types of nephritis.

In this way we succeed in many cases in causing a considerable sweat, so that the patient loses several pounds in weight at each pack, and an existing dropsy may sometimes be made to disappear completely in a comparatively short time. On the other hand, however, we cannot deny that it is very hard sometimes to make patients sweat, even when there is œdema of the skin, and

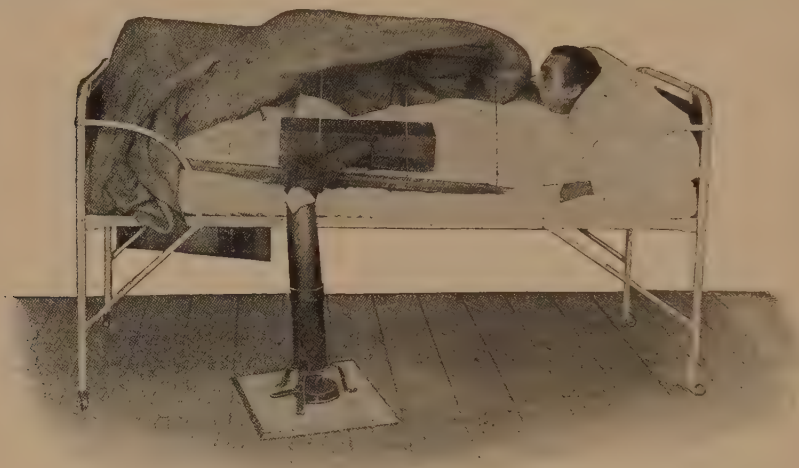


Fig. 98.—Simple hot-air apparatus for diaphoresis. (Erlangen Medical Clinic.)

also that many patients do not bear these sweating treatments at all. The latter is especially true if the patient has dyspnœa, and if signs of cardiac weakness have already set in. In such cases we should employ merely warm baths, brief packs, or hot alcohol rubs.

Of internal diaphoretics there is still one remedy to be mentioned, the hydrochlorate of pilocarpin, prepared from jaborandi. This is best employed subcutaneously, $\frac{1}{6}$ to $\frac{1}{3}$ gr. at one dose (gm. 0.01 to 0.02), but the drug may also be given internally in pills in the same dose. Its effect consists in the production of a rather profuse perspiration, and likewise a marked increase in the salivary secretion, which latter is often a great annoyance to the patient. For this reason, and because it sometimes induces cardiac weakness, pilocarpin is, on the whole, scarcely to be recommended. We ourselves employ it only occasionally when the hot baths are not used or when they do not have a satisfactory effect.

Next to the skin, the intestinal mucous membrane is the organ from which we may soonest expect to produce a vicarious elimination of water, and also of urea. It is sometimes, therefore, of a certain service to prescribe drastic cathartics in nephritis with a diminished secretion of urine, especially if there is a tendency to constipation, besides the dropsy, dyspnœa, etc. The drastic cathartics chiefly used are infusion of senna, extract of colocynth, gamboge in 2-gr. powders (gm. 0.1), tamarinds, Epsom salts, etc.

Finally, it may be asked whether we should not excite the secretory function of the kidneys themselves by the exhibition of diuretics. Certainly the employment of vigorous diuretics is opposed by the consideration that we

might still further irritate and consequently damage the renal epithelium. The milder diuretics, particularly the acetates of potassium and of sodium, in doses of about 1 to 2.5 drachms (gm. 5 to 10) in the course of twenty-four hours, are usually well borne and are often efficient. From diuretin in doses of gr. vijss. to xv (gm. 0.5 to 1) thrice daily, and the related remedies, agurin, theophyllin, we have often seen good results. On the other hand, we would warn against the use of calomel, as very disagreeable symptoms of mercurial poisoning, such as stomatitis, are apt to occur.

If extreme dropsy exists, which cannot be reduced by the above-mentioned methods, and which is very distressing to the patient, the attempt can be made to remove the dropsical fluid directly by mechanical means from the subcutaneous connective tissue. For this purpose we usually use the so-called Southey or Curschmann capillary trocars. These are inserted into dependent portions of the dropsical skin, and fastened with adhesive plaster; the fluid gradually trickles away. In this way several quarts of œdematous fluid can be withdrawn in twenty-four hours, and great relief often given to the patient. Instead of the capillary trocars, some physicians recommend simple scarifications of the skin made longitudinally in the dependent parts (scrotum and posterior surface of the thigh and calf). From these the dropsical fluid is allowed to escape into pads made of peat (gauze), or similar substances. Absolute antisepsis and asepsis are demanded, for there is a great tendency to erysipelatous inflammation.

When uræmia is threatening, and often even when it has broken out, we may try to produce an elimination of the injurious products of tissue metamorphosis from the body in the well-known ways above described, by sweating or drastic purgatives. Besides this, the uræmic symptoms, however, often demand a special symptomatic treatment. If very violent and frequent uræmic convulsions appear, we consider it advisable to try to suppress the attacks by chloroforming the patient. At any rate, it seems to us to be better to use chloroform in uræmia than to give narcotics internally, because with this we can watch the action of the remedy better, especially with regard to the pulse and respiration. Chloroform is also generally used by the obstetricians as the main remedy in the eclampsia of pregnancy. If the attacks are not very frequent, but if there is marked somnolence or coma, tepid baths with cold shower baths are often employed with distinct advantage. Cool baths are also serviceable when there is a great increase of the temperature. If we are treating a robust individual with a full pulse, and during severe uræmia there is a decided redness or cyanosis of the face, venesection may be indicated. This sometimes has a striking and instant effect, as has lately been confirmed by various observers. Great attention is to be paid to the condition of the heart. As soon as the pulse becomes small and weak, energetic stimulants, such as strophanthus or subcutaneous injections of camphor, must be used. If the signs of cardiac weakness appear before the beginning of severe uræmic symptoms, digitalis preparations (digalen, etc.) must be used. Through their action in raising the blood pressure—it being advisable under some circumstances to combine acetate of potassium with them—free diuresis sometimes comes on, and with it a disappearance of the danger from uræmia; but meanwhile we must watch for symptoms of the toxic action of digitalis. The tincture nervina Bestucheffii [nearly equivalent to the tincture of the chlorid

of iron, U. S. P.] may also be sometimes used to advantage in uræmia. We are not apt to interfere with uræmic vomiting or uræmic diarrhea, because these symptoms, as we have said, are to be regarded as a form of self-help by the organism. Only when such symptoms are very distressing do we give cracked ice, morphin, opium, cocain, chloroform, etc. If the vomitus contains ammonia, it is a good plan to give 10 or 15 drops of dilute hydrochloric acid in water several times a day. An abundance of liquids is always to be prescribed upon the appearance of uræmic symptoms, because in this way it is probable that the poisonous blood is diluted and also the excretion of the toxins promoted. We would also briefly mention here that in severe nephritis with lasting anuria and threatening or already developed uræmia (especially in the eclampsia of pregnancy), the bold attempt has repeatedly been made to free the swollen kidney tissue from its internal pressure, and thus to restore the suppressed urinary secretion by operative splitting of the kidney capsule and decapsulation of the kidney. This therapeutic measure has as yet received little support, although a number of surprisingly good results have been obtained. At any rate the ingenious suggestion of Edebohls deserves further consideration.

In severe cases the patient's dyspnœa often demands prompt relief. If the dyspnœa be caused, or at least increased, by hydrothorax, and we do not succeed in removing the hydrothorax in any other way, it is necessary to evacuate it by puncture. In acute nephritis, indeed, we may hope by this means sometimes to preserve the patient's life until improvement sets in. Great ascites must also be relieved by tapping. Against "renal pneumonia" our remedies are powerless. Tepid baths, shower baths, and wet packs sometimes procure relief. In "uræmic asthma" morphin injections may act beneficially. If pulmonary œdema ensues, the heart again is chiefly to be considered. We may try, besides the remedies already mentioned, large mustard plasters, baths, and acetate of lead.

We accordingly see that many remedies are at our service in the treatment of nephritis, the choice of which in the individual case must be committed to the personal judgment of the physician. In the main, we should always begin with the necessary hygienic measures, and, if possible, with a methodical diaphoretic treatment, and govern ourselves otherwise by symptomatic indications. After recovery has set in, great caution is still necessary for a long time. The patient must guard against physical overexertion, errors in diet, and exposure to cold. Preparations of iron are to be prescribed when there is a secondary anæmia.

In regard to the influence of the onset of an acute nephritis on the treatment of the primary disease, we may mention that cold baths are in general not to be freely used, as in typhoid fever with nephritis, but still they may be tried if they are otherwise urgently desirable. We would also suggest that certain internal remedies, especially calomel and salicylic acid, must be used only with great caution when there is nephritis.

CHAPTER III

THE SUBCHRONIC AND CHRONIC FORMS OF NEPHRITIS, WITH THE EXCEPTION OF THE GENUINE CONTRACTED KIDNEY

(Chronic Diffuse Nephritis, Chronic Parenchymatous Nephritis, Chronic Hemorrhagic Nephritis, Large White Kidney, Secondary Contracted Kidney)

Ætiology.—While the acute nephritis described in the preceding chapter runs its course in several days or weeks, and only rarely extends over some months, we will now speak of inflammatory degenerative affections of the kidneys which last at least several months, and often go on for a year or two. The term "subacute" or "subchronic" is chosen for the cases that last a comparatively short time. As we must once more repeat, there is no sharp limit in this respect.

In regard to the ætiology of these forms of nephritis, they do arise from an acute nephritis, but this is quite rare. Formerly such an origin was erroneously regarded as the rule, and this is the reason why the changes in the kidney in these cases were described as the "second stage of Bright's disease" (Frerichs). The English clinical observers Wilks and Johnson, whom Bartels followed in Germany, first pointed out the fact that in most cases the disease shows a chronic character from the start, and that we can only exceptionally, as after scarlet fever, recognize an acute "first stage." The name "chronic parenchymatous nephritis," since frequently used, is chosen entirely from practical reasons, inasmuch as it briefly states the distinction from the genuine contracted kidney; but it is incorrect in principle, as will be shown from the description of the anatomical conditions later. It is more correct, therefore, to employ the term "chronic diffuse nephritis," because we have, in fact, to deal with what is essentially a chronic universal or diffuse disease of the kidneys, as contrasted with the "contracted kidney" in which there are always bits of normal tissue interspersed between the numerous separate foci of disease.

If we look for the ætiological conditions in cases that have a chronic course from the beginning, we can often discover nothing definite at all. The disease seems to have developed "of itself" in previously healthy persons. Most probably we have here some toxic or infectious agency that acts on the kidneys, whose detection, however, is at present impossible. A certain importance seems sometimes to belong to frequent exposure to cold and wet, to damp dwellings, and the like. That hard drinkers, particularly beer drinkers, are especially liable to nephritis seems to the author indubitable, because of his personal observations (*vide supra*). Of course, one will always inquire particularly about previous attacks of acute nephritis, and about other diseases (tuberculosis, syphilis, malaria); but still it is in only a small number of cases that a definite ætiology can be demonstrated.

Persons in early middle life are most frequently affected by the disease, and men more often than women. In children and old people the disease is quite rare.

Pathological Anatomy.—There is no essential distinction between the anatomical lesions of the kidney in acute and in chronic nephritis. The changes

that are seen in both are essentially the same, only they develop and extend more slowly in the chronic forms; and they also, during their longer duration, lead to certain sequelæ in the kidney, which cannot develop at all in acute nephritis, owing in part to the lack of time. Even in chronic nephritis the individual cases differ from one another in many respects. First this and then that histological process is especially prominent, and thus lends certain peculiarities to the macroscopic appearance of the kidneys. Certain sequelæ—such as contractions—have also developed but little in many cases that soon end fatally, but they develop far more in other cases of longer duration. Hence it happens that we can quite well regard certain anatomical forms that are more frequently observed as types, although we must never lose sight of the important fact that there is a pathological unity in all these forms and types. Then we shall not lose the clew to the understanding of the morbid process if the individual case does not always harmonize with the scheme of the text-books.

We distinguish the three following chief anatomical types of subchronic and chronic nephritis:

1. Chronic Hemorrhagic Nephritis in the Form of the Large Red or Variegated Kidney.—The kidney is at least of normal size, and often a little or a good deal enlarged. It feels firmer than normal; its capsule is often adherent to the surface in some places. The surface looks either uniformly a more gray-red or more mottled, while dark-red spots alternate with lighter gray or even yellow spots. The red spots on the surface cannot be wholly wiped off, and thus they prove to be hemorrhages. The gray or yellow parts correspond to the anæmic or fatty-degenerated portions. On section, the cortical substance is usually wider than normal, its normal boundary is obliterated, and its color is a uniform gray-red, or mottled and striated.

Under the microscope we find in part the same changes as in acute nephritis—parenchymatous and fatty degeneration of the epithelium, casts or hemorrhages in the uriniferous tubules, inflammatory œdema or granular infiltration of the interstitial tissue, the capsules of the glomeruli sometimes thickened, the epithelium of the glomeruli sometimes proliferated or desquamated, etc. The special characteristic of this chronic form, in contrast with acute nephritis, is that in many places a complete destruction of the uriniferous tubules has occurred, and that a genuine interstitial connective tissue, more or less rich in cells, has taken their place. In this lies the anatomical evidence of the longer duration of the disease, since the two processes—both the complete atrophy of the epithelium, and especially the secondary proliferation of connective tissue—of course need a certain time for their development. The atrophy and the consequent proliferation of connective tissue usually predominate in some parts, while in others nothing but fresher inflammatory and degenerative changes are perceived.

2. The Inflammatory Fatty Kidney, or the Large White Kidney (*yellow* would be more exact).—In this form of chronic nephritis the kidney is usually enlarged, or at least of normal size. Its outer surface is smooth and of a yellow or an alternating yellow and gray-yellow color throughout. The broader cortical substance shows a yellow and usually somewhat mottled appearance, while the pyramids almost always appear considerably reddened. Hemorrhages are also almost always present in this form, usually, of course,

in smaller numbers than in the variegated kidney, but they are sometimes quite abundant, as in the hemorrhagic fatty kidney.

The microscope shows the great affinity between this form of nephritis and the preceding. We have almost precisely the same changes, including always a partial destruction of renal tissue with a subsequent increase of interstitial connective tissue. The macroscopic appearance of the kidney is due to the fact that it is anæmic, and that the fatty degeneration preponderates in the epithelium. It is worthy of note that in these kidneys marked changes in the glomeruli are usually present.

3. The Secondary Contracted Kidney.—While in the two forms of nephritis thus far described the outer surface of the kidney is still smooth, and the kidney, on the whole, is somewhat enlarged, we have to do here with kidneys of about normal size, on whose surface there are granulations, which as yet are slight, but which still are already plain. This granulation signifies nothing more than that the destruction of the renal tissue has here advanced farther, and that the newly formed connective tissue has in part undergone cicatricial contraction. These kidneys, therefore, represent a later stage of the two forms first named. They usually come under observation when the nephritis has lasted about a year and a half or two years, or even somewhat longer. The first beginnings of granulation may, of course, show themselves earlier, while, on the other hand, when the process lasts a longer time, a completely contracted kidney may develop.

The color of these kidneys is usually reddish or mottled, the red spots corresponding to the sunken atrophic parts, and the gray or yellowish spots to the elevated parts. Yellow kidneys, however, may also show at times decided granulations. Microscopically, we find already marked atrophy of the renal parenchyma, with a corresponding increase of the interstitial connective tissue.

Formerly these kidneys were called the "transition between the second and third stages of Bright's disease." As follows from the above, they are to be regarded only as a more advanced form of chronic nephritis. Since the kidneys, in spite of their granulation, have, on the whole, a normal size, we can infer from this, and from the clinical course, that they were previously enlarged. Therefore the name of "secondary contracted kidney" is quite suitable, in opposition to the genuine contracted kidney, which represents a much more chronic form of renal atrophy. In the latter the contraction takes place in an extremely chronic manner in a kidney which was previously normal, while in the "secondary contracted kidney" the cicatricial process develops in a kidney which was previously the subject of diffuse disease.

Of other pathological lesions, apart from the changes in the kidney, we will mention here only the hypertrophy of the left ventricle, which is found (*vide infra*) in all the above-mentioned forms of nephritis. The chronic parenchymatous nephritis without cardiac hypertrophy, assumed by Bartels in his time, does not exist. Such cases were probably instances of unrecognized amyloid disease. Exceptionally it may happen that when a patient with chronic nephritis is very much debilitated and enfeebled, the cardiac hypertrophy is not developed.

Clinical History.—Only in the comparatively rare cases when the renal affection begins acutely, do the symptoms of chronic nephritis follow immediately on the first acute stage. In most cases, however, the disease develops

slowly and gradually from the start, as we have said, so that it is usually impossible to determine accurately the moment when the disease begins. Still, we may assume that the onset is decidedly more rapid in chronic diffuse nephritis than in genuine contracted kidney.

The first signs of the disease consist of certain general symptoms—pallor, dullness, loss of appetite, nausea and headache, and later of œdema. The latter is often the first symptom which sends the patient to the physician, since in the beginning he is apt to pay little attention to the symptoms first named. The œdema usually appears first in the ankles and legs, more rarely at an early period in the face. It often disappears at first after a night's rest, but always develops afresh during the day, gradually increasing in intensity. The patient himself now sometimes notices a change in the urine, either an abnormal color or cloudiness or a diminished amount. The accurate examination of the urine by the physician first establishes the diagnosis.

In regard to the more special symptomatology of chronic nephritis, we meet exactly the same symptoms as have been described in the preceding chapter on acute nephritis. The characteristic distinction is based merely upon the whole course of the affection and the order of development of the different symptoms, and not upon the symptoms themselves.

The urine almost always is diminished. Of course the figures vary considerably both in different cases and at different times in the same case. A small amount of urine, 10 to 25 ounces (300 to 700 c.c.) a day, is almost always an unfavorable sign, while a free diuresis signifies an absorption of the dropsy and an improvement of the condition. There is also a persistent increase in the amount of urine when the chronic nephritis passes into a secondary contracted kidney. Under such circumstances the daily amount of urine may rise to 1,500 or 2,000 c.c. or more.

The specific gravity of the urine is often increased to about 1.015 to 1.025, corresponding to the amount of albumen and of other solid constituents. It is, of course, correspondingly lower when there is a more abundant elimination of water by the kidneys.

The amount of albumen in the urine is quite marked in all severe cases, being one third to three fourths of its volume. It amounts to about one and one half to three per cent by weight, so that the patient's daily loss of albumen may reach 0.5 to 1 ounce (gm. 15 to 30).

The examination of the sediment, which is usually abundant, is of the greatest importance for the accurate determination of the form of the anatomical changes in the kidneys. Above all, the question arises as to the presence or absence of blood in the urine. If abundant, hæmaturia may be recognized by the naked eye from the color of the urine. The detection of smaller amounts of blood can be made only by the aid of the microscope. It goes without saying that the amount of blood in the urine varies considerably in the different cases, and in the same case the urine often contains much more blood during certain periods in the course of the disease than at other times. The portions of urine passed at different times taken separately often show quite marked variation in this respect; the day's urine usually contains more blood than the night's. From the detection of renal hemorrhages, of course in connection with other symptoms, we can always make with certainty the diagnosis of a "chronic hemorrhagic" nephritis.

In most cases casts are quite abundant in the sediment of the urine, but naturally their amount and variety undergo very great variations in different cases and at different times in the same case. They are the direct sign of the presence of an inflammatory exudative process in the kidneys, although the deposits on the casts are more important for the diagnosis of the special form of renal disease than are the casts themselves. Those formed constituents of the sediment are most characteristic in this respect which point directly to the processes of fatty degeneration in the kidneys: the fatty granules and fatty granular cells, free or attached to the casts. The number of these elements is especially great in the chronic inflammatory fatty kidney, the "large white kidney." The usually clear, nonhemorrhagic urine may in some cases have even a fatty lustrous surface. Renal epithelium is, on the whole, more rarely present in the sediment in chronic nephritis than in acute, but it occurs in some cases.

Of the other symptoms, the one that usually most strikes the eye is dropsy. It usually comes on, as we have said, in the beginning of the disease, and slowly or rapidly reaches a great extent and intensity. A medium or even a high degree of general dropsy may often persist almost unchanged for months. In other cases it shows either spontaneous variations or variations influenced by treatment; it decreases for a time only to increase anew. The severer and more comparatively acute the case, the greater in general is the dropsy. In the more chronic cases, in secondary contracted kidney, its intensity may be slight for a time or even permanently. The dropsy may even be absent in some cases, as we learn especially from the observations reported by Wagner under the name of "chronic hemorrhagic Bright's disease without œdema" (*vide infra*). In regard to the different localizations of the dropsy, and to dropsy of the internal cavities, hydrothorax, ascites, and hydropericardium, and their results, the same holds true as in acute nephritis.

Of the internal organs, the condition of the heart lays claim to the most interest. In all cases of chronic nephritis in which we do not have to do with especially weak and run-down patients, who cannot save the necessary nutritive material for the formation of a cardiac hypertrophy, we find a pronounced and often a very marked hypertrophy of the left ventricle, either with or without a coexisting dilatation of its cavity. The detection of cardiac hypertrophy during the patient's life is sometimes difficult, especially when there is general dropsy, but the diagnosis can usually be correctly made with proper attention to the abnormally tense radial pulse, the accentuated, valvular aortic second sound, and the displacement outward of the apex beat, or at least its increased strength. We often find in the cadaver, and can sometimes make out during life, a hypertrophy of the right ventricle (*vide supra,* page 742).

A second important sequel of chronic nephritis consists of the changes in the retina—albuminuric retinitis. Although very rare in acute nephritis, these changes are present in the majority of the cases of this class. Sometimes the patient's subjective visual disturbance (dimness of vision, defects in the field of vision) points to a disease of the retina, but the existence of disease can be established with certainty only by ophthalmoscopic examination. The albuminuric retinitis usually appears on both sides. It generally begins with a mild optic neuritis; then in most cases we find, first, retinal

hemorrhages, and, second, white spots and streaks, especially in the vicinity of the optic nerves. The origin of the spots, which may appear and disappear again, is not yet entirely clear. At any rate, they are circumscribed fatty degenerations of the special retinal elements. The degree of amblyopia depends, of course, chiefly upon the localization of the changes, whether in the macula lutea or other parts.

We need say little in regard to the other symptoms, since they agree essentially with those of acute nephritis. The general anæmia is very pronounced in many cases, but it is less marked in the very chronic forms. The cerebral symptoms, especially the headache and the mild vertigo, may depend in part upon the cerebral anæmia; otherwise they are due to uræmia (*vide infra*). Cerebral hemorrhages have been observed in a very few cases. Hemorrhages on the inner surface of the dura mater are more frequent, but they are usually without clinical significance. The mouth, larynx, and pharynx usually show nothing particular, except accidentally complicating inflammations. We must, however, remember the occasional occurrence of a very distressing or even dangerous œdema of the soft palate, or of the arytænoepiglottic ligaments—œdema of the glottis. Forms of bronchitis and pneumonia occur similar to those seen in acute nephritis. Bronchitis and chronic œdema of the lungs also make their appearance in the more advanced stages of the disease, as a result of cardiac insufficiency. Finally, we must remember the hindrance to respiration from hydrothorax, and also from uræmic dyspnœa. The changes in the heart have already been spoken of. Endocarditis or pericarditis may occur, but they are very rare.

As to digestive disturbances, loss of appetite is a very common symptom. Very persistent vomiting is usually to be regarded as a chronic uræmic symptom. The bowels, as a rule, are constipated, but there may also be severe diarrhea, as in acute nephritis. In severe cases, especially in the last stages of the disease, ulcerative and dysenteric processes have repeatedly been observed in the large intestine and the ileum. Peritonitis may occur, but it is at all events extremely rare. The liver and spleen usually show no peculiarities.

Uræmic symptoms, both of the milder chronic variety and also in their severest acute form, may come on at any time, although they do not by any means attain their full development in all cases, and are somewhat rarer than in genuine contracted kidney.

The temperature remains normal, as a rule, as long as it is not influenced by complicating inflammations, or by the appearance of uræmia.

Course, Duration, and Termination of Chronic Nephritis.—In general, the whole course of chronic nephritis presents quite a great uniformity. The different symptoms may show certain variations within long periods, but the patient often presents almost the same appearance day after day for months. The duration of the disease varies greatly, from three to six months, in the subacute cases, to two or three years, or even more, in the very chronic cases. The cases of long duration are almost all cases of secondary contracted kidney. They sometimes show in their clinical relations the transition from the enlarged to the granular kidney, since the picture in many of its details is more like that in the genuine contracted kidney: the œdema decreases, disappears completely, or at least continues in a lesser degree; the amount of

urine becomes more abundant, and the specific gravity and the amount of albumen become correspondingly less. The condition thus lasts for a long time until it grows worse again, through uræmia or disturbance of the compensation in the heart.

The final termination of chronic nephritis is in most cases unfavorable. In the severe forms death ensues in from three months to a year, either in consequence of general dropsy or from uræmia, from complicating inflammations, etc. The conditions when the nephritis goes on to secondary contraction are comparatively more favorable, inasmuch as the patient may then find himself in a tolerable state, for a time at least. Complete recoveries doubtless occur in chronic nephritis, but they are rare. The longer the disease lasts beyond the first six months the less likely is recovery. The development of secondary contraction may simulate recovery, or, at any rate, the subjective condition of the patient may be almost perfect. Even after signal improvement, however, relapses are always to be feared. There are even genuine acute attacks in the course of chronic nephritis.

Different Forms of Chronic Diffuse Nephritis. Diagnosis.—By means of careful examination of the urine in all suspicious cases—for example, those presenting œdema, anæmia, and similar symptoms—we may in general be certain of making a correct diagnosis of chronic nephritis, but all experienced observers will acknowledge that the diagnosis of the particular form of chronic nephritis is difficult and uncertain, even when the examination of the case is most painstaking. Still, the following schematic *résumé* may at least furnish some indications:

Chronic Hemorrhagic Nephritis (large variegated or mottled kidney).—Duration from six to eighteen months. Urine hemorrhagic; usually quite rich in red blood corpuscles and casts. Œdema. Cardiac hypertrophy. Retinal changes. Quite frequently uræmia.

Inflammatory Fatty Kidney (large white kidney).—Duration also six to eighteen months, but usually somewhat shorter than in the preceding form. Urine not at all, or only slightly, hemorrhagic. Frequently many white blood corpuscles, and especially signs of fatty degeneration in the kidneys, fatty granular cells, fat drops in the urine, etc. Large amount of albumen in the urine. Marked œdema. Cardiac hypertrophy. Very often retinal changes. Death by uræmia frequent.

Secondary Contracted Kidney.—Longer duration of the disease, from a year and a half to three years. At first the symptoms of the preceding forms; later, urine more abundant, less œdema, etc. Death from an increase of the dropsical symptoms due to cardiac insufficiency, uræmia, etc.

Chronic Hemorrhagic Nephritis without Œdema.—This form has been already mentioned (page 767), but it should receive somewhat further consideration. Up to this time it has been too little regarded, although we ourselves have observed not a few cases of this variety of chronic nephritis. The disease has a rather chronic course. For a long time the patient is but slightly inconvenienced, particularly if he has good care. The urine is usually quite abundant, and contains very little albumen, but has always, or almost always, small amounts of blood mixed with it, and this continues for many months or even longer. From time to time there will appear a more or less considerable hæmaturia, so that the urine has an abundant dark, brownish-red sediment.

This is especially likely to occur if the mode of life has been indiscreet, but it may also happen without exciting cause. The urinary sediment is composed of red blood corpuscles, some of which are entire and some already disintegrated together with a moderate number of hyaline casts. The latter usually have red blood globules or granules of hematoidin and detritus adherent to them. The amount of urine temporarily diminishes, but it soon increases again. Little, if any, increase in the size of the heart can be demonstrated; œdema is entirely absent, nor have we yet observed retinitis in association with this form of nephritis. Uræmia does occur, but it is rare. The course of the disease is very tedious, as we have said. Whether recovery may take place is uncertain. Usually apparent recovery is followed by the sudden appearance of a fresh hemorrhage. It is probable that in most cases the final result is a secondary contracted kidney.

With regard to the ætiology of this form of chronic nephritis, it is our opinion that in most cases there is a chronic septic infection or intoxication; at any rate, it is noticeable that such patients not very infrequently give a history of some suppurative process.

Treatment.—The treatment of chronic nephritis corresponds in all its details so closely to that of acute nephritis that we can refer almost entirely to the preceding chapter.

The main thing here also is regimen and symptomatic treatment. The patient must always keep himself warm, wear flannels, or stay in bed. The diet must, in general, conform to the rules prescribed in the discussion of acute nephritis. For a time we may try a diet in which milk predominates. A diet lacking in salt is particularly indicated in the cases with a tendency to œdema. It is always advantageous to give an abundance of liquids, except that alcoholic beverages should be avoided so far as possible. It is well to pay careful attention to the skin—bathing, or sponging with warm water and alcohol. Under some circumstances climatic treatment, in Italy, Egypt, etc., are indicated in the more chronic forms. We see good results occasionally, in particular, from the warm, dry climate of Upper Egypt; but we must not expect too much of this kind of treatment, which generally involves great sacrifices.

The treatment of dropsy follows entirely the methods previously described, and so does the treatment of any uræmic symptoms. If there is persistent hæmaturia, ergotin may be tried, but it seldom seems to do any good.

If the graver symptoms of nephritis are absent, we confine ourselves to a general dietetic treatment, prescribing warm baths, massage, and, if anæmia be present, preparations of iron. In cases of this character, such baths as Brückenau, Wildungen, and sea baths may be recommended.

CHAPTER IV

CONTRACTED KIDNEY

(Genuine Contracted Kidney. Granular Atrophy of the Kidney. Granular Kidney. Chronic Interstitial Nephritis)

Definition and Ætiology.—The genuine contracted kidney is the result of an extremely chronic and very slowly but constantly progressive atrophy of the

renal tissue. The term " chronic nephritis " is also used for contracted kidney, but the special inflammatory processes are very subordinate here, for the anatomical change consists essentially in nothing but a simple degenerative atrophy of the renal parenchyma, and in a corresponding gradual increase of the interstitial connective tissue. From a general pathological point of view the process is to be regarded as wholly analogous to the corresponding changes in the liver in cirrhosis of that organ, in the spinal cord in the chronic degenerations of the different systems of fibers, etc. In all these cases we have a primary destruction of the special tissue elements as a result of some deleterious action, and, following a general pathological law (Weigert), a partial replacement of the parts destroyed by a newly formed cicatricial connective tissue.

In the " genuine " contracted kidney the atrophy of the renal parenchyma begins in a previously healthy kidney. Cell after cell of epithelium, islet after islet of tissue, are slowly attacked, while other parts still remain intact. It was therefore an error of the older pathologists to regard the contracted kidney as the " third stage of Bright's disease," as if every granular kidney were first found in the stage of acute inflammation, and then passed into the stage of chronic enlargement, and lastly into that of contraction. This theory, of course, suits certain cases in part, for chronic nephritis at least may often finally pass into contraction, but these " secondary contracted kidneys " (*vide supra*) can clinically, and almost always anatomically, be differentiated from the genuine contracted kidneys. The contracted kidney may arise from an acute nephritis in some cases, and careful investigation indicates that such cases are not very rare; but then the process hardly ever passes through the three stages mentioned above, for the acute nephritis apparently recovers. A slight remnant of it is left—a little fire, as it were, smoldering under the ashes; its work of destruction advances, wholly in secret, and perhaps only after many years do the symptoms of a pronounced renal contraction appear.

If we inquire into the causes which produce the atrophy of the renal tissue in the ordinary cases of contracted kidney, which are chronic from the first, we are very often unable to make out any special ætiology. Of course, one of the first things to consider in this disease is chemical or toxic agents, whether these are introduced directly as such into the body, or whether they are manufactured in the body because of abnormalities in metabolism, or as a result of infectious processes.

Experience teaches us that there are three chemical substances to be mentioned which may favor the development of contracted kidney: alcohol, lead, and uric acid. Chronic alcoholism is often to be regarded as the most probable cause of renal contraction, especially in people who have " lived well " otherwise, and have become corpulent. In these cases contracted kidney is to be viewed as a sort of atrophy, due to wearing out, or to strain. It is difficult to decide how much influence should be ascribed to the alcohol itself and how much to the excessive ingestion of food. The specific effect of alcohol is almost indubitable in those cases in which contracted kidney and hepatic cirrhosis are both present, a combination repeatedly observed. The connection between contracted kidney and chronic lead poisoning, in typesetters, painters, etc., is also incontestable. It is a remarkable circumstance, and one not yet fully explained, that in these cases we very often see at the same time genuine gout (*arthritis uratica saturnina*). If we make careful inquiries into the

history and previous symptoms of the patient, almost invariably we will be able to determine that previously attacks of genuine articular gout have occurred. Gout, however, alone, without any coexisting chronic lead poisoning, often leads to the development of contracted kidney, "gouty kidney," in which we probably have to do with the noxious action of an abnormal amount of uric acid on the renal parenchyma; but it always seems to be especially unfavorable to have an accumulation of several injurious factors—for instance, lead poisoning associated with chronic alcoholism.

Infectious influences are, probably, first to be considered in those cases where the contracted kidney can be referred to a former acute infectious nephritis, as after scarlet fever. We may also mention here the appearance of contracted kidney sometimes observed after severe acute articular rheumatism. We may perhaps imagine a similar connection in the cases where contracted kidney is found combined with chronic endocarditis (valvular heart disease), or with chronic arthritis not of gouty origin. Of the chronic infectious diseases, which sometimes have a connection with the origin of contracted kidney, we may mention malaria and syphilis. The latter ought especially to be considered more than it is at present, because we may have either an immediate action of syphilitic toxins, or a renal atrophy as the result of a specific disease of the renal arteries.

We must here devote a little time to a consideration of the connection between contraction and primary disease of the vessels, which has been much discussed. It is true that we often find general arteriosclerosis, and also atheroma, especially in the renal arteries, in the bodies of persons who have died from contracted kidney, but this frequent coincidence cannot be remarkable in such cases, because contracted kidney is seen chiefly in elderly persons, and those in whom atheroma of the arteries is also a very common symptom. The theory advanced by the English authors, Gull and Sutton and others, that the vascular disease, "arteriocapillary fibrosis," always represents the primary process, to which the renal atrophy is only secondary, is, however, utterly untenable. We often find the most pronounced contraction of the kidneys without any vascular changes sufficient to explain the atrophy; and where the latter can be found in the small renal arteries, we usually have not a primary but a secondary process—namely, the well-known obliterating arteritis, which is seen in almost all chronic inflammations and degenerative atrophies of various organs.

Of course, it cannot be denied that primary disease of the arteries in the kidneys may check the flow of blood to certain portions of the tissue, and thus occasion secondary atrophy in limited areas (arteriosclerotic contracted kidney), just as, for example, interstitial myocarditis results from primary arteriosclerosis of the coronary arteries. This is particularly true of the so-called senile kidney—that is, the granulated kidney of old persons, due to atheromatous changes in the blood vessels, and perhaps also certain other cases of nephritis subsequent to syphilis, and especially the rare cases of unilateral contracted kidney, which have been seen chiefly in association with syphilis.

Finally, we have yet to call special attention to the fact that we not infrequently observe cases of contracted kidney, more particularly in younger persons and in women, in which the most careful investigation fails to disclose any causal factor, and the disease appears to develop altogether spontaneously.

We are inclined to ascribe these cases, at least in part, to a congenital lack of power of resistance in the tissues of the kidney. Kidneys of this character are not equal to the ordinary functional demands on them for any length of time. In a certain sense we may well compare such cases of "primary progressive atrophy" of the kidneys to "progressive muscular atrophy"; all the more readily as our own experience shows that a family or hereditary predisposition can sometimes be made out in such cases of renal disease. Even in cases in which it is certain that extraneous causes are involved, we must always take into consideration the individual predisposition and the power of resistance of the tissues.

With relation to the age and sex of patients with contracted kidney, it has already been repeatedly stated that the disease occurs mainly in later life. The explanation of this fact lies in the peculiar circumstances which occasion the renal process. For the same reason, also, the male sex is much oftener attacked than the female; yet the causes which lead to contracted kidney are so numerous that it is easy to understand why the disease is not infrequently seen even in younger individuals and in women. Contracted kidney does occur in children, though rarely. In all cases in youthful persons we must make careful inquiry as to any previous attack of acute nephritis, subsequent perhaps to scarlet fever, diphtheria, or measles.

The relations of contracted kidney to amyloid disease of the kidney (so-called amyloid-contracted kidney), and to chronic disease of the urinary passages, particularly of the pelvis of the kidney, will be discussed later in the appropriate chapters.

Pathological Anatomy.—In the genuine contraction of the kidney, both kidneys are always diminished in about the same degree. Their size is sometimes reduced to one half or even one third of the normal, so that it is almost difficult to find the little kidney in the very abundant and thick fatty capsule that is often present. The kidneys feel firm and dense, and show on their surface a very plain, coarse or fine, uniform or irregular, granulation. On pulling off the somewhat thickened fibrous capsule, these granulations become more prominent, and the capsule usually adheres quite firmly to the depressed portions. The raised portions are almost always darker and redder—that is, richer in blood—than the lighter and grayer depressions. Whether the whole kidney appears more red or more white depends only upon the amount of blood in the organ, and there is no reason for separating the "small red" from the "small white" contracted kidney.

On section of the contracted kidney we find the cortex very thin, and pale atrophic streaks alternating with the darker portions. The pyramids are also rather small, and, as a rule, are darker than the cortex. In the pelvis of the kidney, which is often somewhat dilated, there are sometimes a number of uric-acid concretions. Striated uric-acid infarctions in the pyramids are a very characteristic mark of the gouty contracted kidney. The microscope shows an advanced destruction of the renal parenchyma, which is replaced by a cicatricial connective tissue in which the nuclei are still numerous or else have already become scanty. We can always make out signs of degeneration and atrophy of the epithelium, and the formation of casts in the uriniferous tubules which still remain, but which are already diseased. Atrophy, thickening of the capsule, etc., are found in many of the glomeruli. The uriniferous tubules

that are still preserved in some places are often in part dilated. We cannot here go more fully into the manifold histological details, especially the formation of cysts, the changes in the vessels (*vide supra*), the deposition of lime salts, etc. Hemorrhages are only very rarely present.

Thus the contracted kidney may be regarded as the form of chronic nephritis with by far the longest course (lasting from three to five years, and even much longer), and also the form with the widest extent. Its essential nature can in no way be contrasted with "chronic parenchymatous nephritis" as a "chronic interstitial nephritis"; for we always find interstitial processes in the former, which have reached a far higher degree in the contracted kidney only because the slow atrophy of tissue is compatible with a much longer duration of life, and hence can attain a much greater extent.

The anatomical changes in the other organs of the body besides the kidneys will be spoken of in connection with the symptomatology of contracted kidney.

Clinical Symptoms.—Except in the comparatively rare cases where we can refer the origin of a contracted kidney to a previous acute or chronic nephritis, the clinical symptoms of contracted kidney develop as gradually and insidiously as the anatomical process itself. There is no doubt that a contraction of the kidney may exist for years without calling the patient's attention to his disease by a single serious subjective symptom. This is evident from the chance discoveries on autopsy of a contraction of the kidney in people who have lost their lives in some other way, but especially from the cases where the severest symptoms, such as uræmia, cerebral hemorrhage, etc., which often lead immediately to death, suddenly come on in persons previously regarded as perfectly healthy, while the autopsy shows a quite far advanced contraction of the kidney as the special cause of these symptoms. The less prominent the subjective symptoms of renal contraction are in the earlier stages of the disease, the more we should consider the objective changes, which in fact usually permit the diagnosis of the disease quite early on careful examination of the patient.

The condition of the urine is most important in this respect. As soon as changes have taken place in the epithelium in different parts of the kidneys, the results previously spoken of must make themselves manifest in the secretion of the urine, although still in a slight degree, and the diseased portions will secrete a urine diminished in amount and in solid constituents, but containing albumen. Since, however, many normal uriniferous tubules and glomeruli are still present, and since the whole process, as we have seen, develops only very slowly, the body gains time for the development of one of those apt compensatory arrangements which we recognize in so many pathological processes, and which we must regard in a teleological sense. This compensatory process consists of an increase in the arterial pressure, coming on as gradually as the renal contraction itself, and constantly increasing, and of a hypertrophy of the left ventricle dependent upon it. The blood therefore courses through the many normal glomeruli of the contracting kidney under an increased pressure, and the consequence is that in these portions the secretion of the urine, especially of the water, is much more abundant. It is possible, also, that the incipient disease of the glomerular walls tends to increase their permeability, and thus contributes to the polyuria. At any rate, it is a fact that in cases of contracted kidney there is usually an abnormally large amount of pale, watery urine of low specific gravity, containing merely a trace

of albumen (originating from the diseased portions of the organ). The daily amount of urine is often 70 to 120 ounces (2,000 to 3,500 c.c.) or more; the urine looks light yellow and clear, contains scarcely any morphological constituents, has a specific gravity of 1.010 to 1.005 or even lower, and gives, on heating, only a slight precipitate of albumen, the amount excreted in the twenty-four hours being about 0.5 to 1 drachm (gm. 2 to 5). On careful microscopical examination of the urine, we usually succeed in finding a few hyaline casts, which only exceptionally may be more abundant. The urine also frequently contains some white, and more rarely a few red, blood corpuscles. In rare but definitely attested cases it may happen that for a time, or even during the main part of the disease, the urine contains no albumen at all, or only a trace of it. This is probably explained by the fact that the diseased glomeruli have wholly ceased secreting, and that therefore the urine is secreted only by the healthy portions of the kidney. As to the character of the urine in arteriosclerotic contracted kidney, *vide infra.*

It is apparent of how great significance this abundant secretion of water, as a result of the abnormally high blood pressure, must be for the whole morbid process; for, in spite of the renal disease, there is now absolutely no retention of water in the body, and we therefore understand why there is often no œdema in contracted kidney, even after a course of years. The secretion of the solid constituents of the urine is not quite in such a favorable condition as the secretion of water. It is self-evident that the percentage of the former decreases with the increased amount of urine, but the whole amount of urea, uric acid, phosphoric acid, etc., eliminated is also at times somewhat less than normal in relation to the food. This diminution, however, is not very great, as long as the work of the heart is sufficient, and at certain times, especially in the earlier periods of the disease, a normal amount may be secreted. We accordingly see that the symptoms dependent upon an accumulation of the urinary constituents in the blood do not appear at all for a long time. Thus it happens that the patient may still feel perfectly well up to a time when the objective examination of the urine indicates marked pathological changes. Many patients, of course, notice the polyuria, but often no special attention is paid to it, and it is attributed to drinking a good deal of fluid. The patient gets accustomed to it, even if, as often happens, he has to pass his urine much more frequently than formerly, and even during the night.

We need not go into detail here in regard to the special causes of cardiac hypertrophy (compare page 740). It was with regard to contracted kidney that Traube advanced his mechanical theory of cardiac hypertrophy, which, however, rested upon the considerations previously mentioned, and therefore was properly replaced by the chemical theory, which was also very applicable to this form of renal disease. In its clinical relations it is an important fact that the cardiac hypertrophy causes no subjective symptoms at all as long as the heart can suffice for the work put upon it without strain, a condition which is perfectly analogous to that of any fully compensated valvular disease. We can usually recognize the condition correctly only by a careful physical examination of the heart and the vascular apparatus, although in contracted kidney the percussion and palpation of the heart are often rendered difficult by a coexisting pulmonary emphysema. We can often perceive, however, the displacement and the increased strength of the apex beat, the extension of

the cardiac dullness to the left, and almost invariably the abnormal tension of the radial pulse and the accentuation of the aortic second sound. In the later stages of the disease a hypertrophy of the right ventricle is often added to that of the left (compare page 742). Complete, or almost complete, absence of the cardiac hypertrophy is observed, as we have said, only in weak and cachectic patients.

As long, therefore, as the high arterial pressure kept up by the cardiac hypertrophy regulates the renal secretion in the way above described, the condition of the patient, as a rule, shows no special abnormality. At most it happens that certain cerebral symptoms now appear, especially attacks of headache and occasional vertigo, which, unless they are uræmic, are probably to be referred to active cerebral hyperæmia. Frequent nosebleed also sometimes results from the abnormally high blood pressure.

The clinical picture is completely transformed, however, upon the appearance of the first signs of incipient cardiac insufficiency—that is, the moment that the hypertrophy of the left ventricle ceases to be sufficient to overcome the obstacles to the secretion of urine occasioned by the deficiency in the renal parenchyma. The disturbance is evident either when the left ventricle itself grows weak, or when the gradual advance of the diseased process has reached such a point that even the most vigorous efforts of the heart are no longer sufficient to bring about compensation. In the latter case the symptoms which appear are to be regarded as uræmic, in the broadest sense of that word. The pulse is frequent, but it still remains unusually full and hard. On the other hand, in case there is a diminution in the cardiac energy, the pulse becomes more compressible, smaller, more frequent, and sometimes, toward the termination of the disease, irregular. The heart sounds remain pure, but the first sound often becomes indistinct. When there is a decided disturbance of compensation we hear a well-marked *bruit de galop*. In all these cases the symptoms result from the disturbance to the circulation, and from the consequent retention of urinary constituents.

Ordinarily, the subjective symptoms of contracted kidney begin very gradually, then disappear for a time, then appear again, and grow worse slowly but steadily. Apart from the feeling of general languor and weariness, it is usually the dyspnœa which first calls attention to the disease. The patient grows short of breath, is much disturbed by even slight physical exertion, and is perhaps subject to attacks of palpitation. Not infrequently the dyspnœa occurs, in the later stages of the disease, in distinct paroxysms, which suggest asthma. This nephritic asthma has long been recognized. It does not have the same origin in every case; often it is undoubtedly the result of the attacks of cardiac weakness, and is then a merely cardiac asthma and corresponds in its individual symptoms to the dyspnœa of heart disease. In other cases the asthma seems to be connected with the retention of the products of metabolism (uræmic asthma, *vide supra*). The clinical picture is most peculiar in those cases in which the dyspnœa is associated with the signs of acute pulmonary œdema, and is attended with the expectoration of a large amount of foamy, serous sputum, which is often tinged with blood. These conditions, which may pass off and reappear at stated intervals, are mainly those to which the name of humid asthma was formerly given. It may seem questionable whether we should regard the pulmonary œdema in such cases as a purely

congestive transudation due to cardiac weakness, or as an œdema occasioned by nephritis and in a certain degree inflammatory. In the last stage of the disease there is often constant dyspnœa, and this may be the chief complaint of the patient. It is in such cases often referable to various causes acting simultaneously; for instance, pulmonary congestion, diffuse bronchitis, pneumonia (*vide infra*), and hydrothorax.

Another result of beginning cardiac failure in the later course of the disease may be œdema of various portions of the body. This is certainly in many cases to be regarded as a purely congestive œdema, particularly when associated with contracted kidney; but, on the other hand, we cannot always exclude conditions which occasion nephritic œdema (*vide supra*). It has, indeed, been repeatedly observed that dropsy may be entirely absent in contracted kidney; but this is the case only when death ensues from some intercurrent attack before the pronounced cardiac insufficiency. Otherwise œdema is by no means rare in contracted kidney. It usually appears first in the ankles, the eyelids, or the prepuce, disappears again when the patient remains at rest, and, after a longer or a shorter pause, comes on anew, until finally, in the last period of the disease, a high degree of general dropsy may develop.

Among the disturbances of the internal organs we must mention first the cerebral symptoms. While at first, as we have said, these have more of an active hyperæmic character, the frequent and very violent headaches that come on later in part are of a uræmic nature and in part depend upon the passive hyperæmia, or the arterial anæmia of the brain. The pain sometimes shoots into the back of the neck, and sometimes is localized chiefly in one half of the head; it is often associated with symptoms of vertigo, with a gloomy or morose mood, with troubled sleep, etc. The stasis is usually also apparent in the abdominal organs. Chronic dyspeptic disturbances appear, the appetite fails, the bowels become irregular, and we can even make out a moderate enlargement of the liver. The influence which the altered activity of the heart exerts upon the function of the kidneys themselves is, however, particularly important. From what has been previously said of the dependence of the secretion of urine upon the arterial pressure, it follows directly that any compensatory activity of the still normal renal territory must at once experience a reduction, as soon as the blood pressure falls. Corresponding to this we see, in fact, that the secretion of urine also usually suffers a decline at the same time with the other symptoms of stasis. The amount of urine is less abundant: it drops to 40 or 50 ounces (1,500 to 1,000 c.c.), and even lower; the specific gravity rises, rarely to a high figure, but still up to 1.010 or 1.012, or over. The urine often retains its light color for quite a long time, but it may finally more and more resemble the genuine urine of stasis. The point, however, which is especially to be considered, is the simultaneous and increasing retention of the solid constituents of the urine in the blood, and the consequent possibility of the onset of uræmic symptoms.

It must be stated that, in contracted kidney, the immediate exciting causes of uræmia are not always clear. Thus, it is a well-known and very important fact, clinically, that very severe and often fatal uræmic convulsions may sometimes attack the patient quite suddenly, apparently when in the best of health. Cases have been repeatedly seen, by other observers and by ourselves, where the daily amount of urine has shown no discoverable diminution in the days

preceding the uræmia. Probably the explanation of this is that for a long time very minute amounts of toxic material are daily retained in the blood, and that these of themselves occasion no noticeable disturbance until suddenly the severe symptoms of uræmia develop as a cumulative result of long-continued, though slight, injurious influences. These cases of suddenly developing uræmia always remind us of the similar phenomena in chronic poisoning from lead or mercury. In these cases, also, after a long-continued absorption of trifling amounts of poison, the symptoms of intoxication may finally appear abruptly. In other cases of uræmia associated with contracted kidney, the condition of the heart is of great importance. The uræmic symptoms develop in different ways, according to the slowness or rapidity with which the cardiac insufficiency causes diminution in the urinary secretion. If the amount of urine is slowly diminished, we observe the symptoms of chronic uræmia (page 739), consisting of headache, vomiting, diarrhea, severe pruritus of the skin, etc., but these symptoms are, of course, often combined with the immediate symptoms of stasis, and are not always to be easily and distinctly separated from them. Such a condition of chronic uræmia, in patients with contracted kidney, often presents a very mournful picture, since the unrestrainable and constantly recurring vomiting, the headache, and the general mental anxiety may last for weeks. The severe acute uræmia either is preceded by chronic uræmic symptoms, or comes on at once in the severest form, with general and often-recurring convulsions, and coma. The uræmia may pass off again, even in contracted kidney, but quite frequently it is the immediate cause of death (*vide infra*).

Besides the symptoms so far described, we must now mention also a number of anatomical complications which may appear in the course of contraction of the kidney. From its diagnostic and clinical importance the albuminuric retinitis, already known to us from the preceding chapter, takes the first place. It may come on at any time in the course of the disease; but it often develops so early that the patient, up to this time, knows nothing at all of his other disease. He merely consults an oculist, who often first recognizes, from the ophthalmoscopic image (see page 767), the special seat of the primary disease. Even in the cases where no subjective visual disturbance is present, the retinal examination sometimes discloses retinitis. In general, the contracted kidney is that form of renal disease in which retinal changes are decidedly most frequent.

Another rarer but clinically important complication consists of the hemorrhages in internal organs, whose cause is to be found either in the increased arterial pressure, or in an abnormal weakness of the walls of the vessels—arteriosclerosis in older persons, defective nutrition of the vascular walls in young and anæmic patients. Hemorrhages into the brain are comparatively the most frequent. They cause both mild and severe apoplectic attacks, which pass off completely or leave a hemiplegia behind, and sometimes they are the direct cause of death. Besides the hemorrhages into the brain itself, there may also be hemorrhages on the inner surface of the dura mater—hematoma. Nosebleed is also of significance; in many patients it is frequent and very stubborn; we have ourselves seen two cases where the fatal termination was caused directly by an unrestrainable nosebleed. Hemorrhages into the other organs are more rare, but they have been observed in the skin, the stomach, the intes-

tines, and the lungs. In a few cases, indeed, a sort of hemorrhagic diathesis seems to develop. We must remind the reader again, in this connection, of the general truth that in renal disease all the internal organs display a somewhat increased tendency to secondary inflammation; thus the mucous membranes often present an accompanying catarrhal inflammation, such as chronic laryngitis, bronchitis, gastric catarrh, and intestinal catarrh. These catarrhal troubles are in part to be regarded as due to congestion, but in part they certainly result from the retention of the products of metabolism. In serous membranes we often observe pleurisy, pericarditis, etc. The surface of the body sometimes displays a tendency to obstinate eczema. Of the inflammations of internal organs, pneumonia is the most frequent and important. Some cases are croupous or lobar, and some are of that diffuse lobular variety which is peculiar to all sorts of nephritis. We have already mentioned that the kidney itself may suffer from inflammatory exacerbations, in addition to the chronic disease.

Very great variations appear in regard to the general nutrition. In most cases where the disease develops quite gradually in persons in middle or advanced life, the general nutrition for a long time shows no striking anomaly. The patient is often very well nourished, and even corpulent, at the period when the first cardiac symptoms begin. To the more practiced and attentive eye, of course, he shows a certain appearance of suffering, which later becomes more pronounced. He becomes emaciated, and has a sallow and often cyanotic complexion. Marked anæmia usually develops only in younger individuals, who then show the pallor characteristic of so many patients with renal disease.

We desire to append a few remarks with regard to arteriosclerotic contracted kidney. This may pursue precisely the characteristic course of the ordinary disease, but in many cases its symptoms are very obscure. If we examine carefully, from day to day, the urine of old persons who present other signs of general arteriosclerosis, we shall be likely to find at many times a small trace of albumen, and at other times none at all. In such cases we may be almost certain that there is an arteriosclerotic contracted kidney. Still, the renal symptoms are usually of minor importance in the clinical picture of senile marasmus, senile emphysema, senile dementia, or the other changes of old age.

General Course, Duration, and Termination.—The most important peculiarities in the course of renal contraction have already been spoken of above. We have stated that the disease may be latent for a long time; that the severest symptoms—such as uræmia or apoplexy—sometimes come on suddenly and unexpectedly; that in other cases the disturbances of compensation in the heart, dyspnœa, palpitation, or slight œdema, are the first symptoms; that, under some circumstances, certain complicating conditions, such as retinitis, or frequent nosebleed, first direct suspicion to a renal disease, and suggest an examination of the urine; while, finally, in a last class of cases, only general disturbances, loss of appetite, pallor, general physical weakness, and similar symptoms induce the patient to consult a physician. It is usually hard to decide how long the disease has lasted before a diagnosis is made. We must especially inquire into the existence of polyuria, which patients may not notice, however, even if it exists.

The further course may vary according to the onset of complications, the

external conditions under which the patient lives, etc. In general, as we must repeatedly emphasize, much depends upon the heart's capacity for work and its staying qualities. If death does not ensue sooner from some intercurrent disease, the last stage of the disease almost always presents itself under the picture of cardiac insufficiency with predominant symptoms of dyspnœa and general dropsy.

As has been said, we usually cannot determine with any accuracy the duration of the disease. It may, at any rate, last many years, probably even ten years or more, although there may be many variations in its course. It is not impossible that, during the earlier period of the disease, there may be a cessation in the process of renal atrophy, but it is hard to decide with certainty. At all events, the disease must generally be termed absolutely incurable, although life may not only be preserved for a long time, but the patient may even exist without much discomfort. We need not refer especially here to the different intercurrent attacks, the possibility of which must always be kept in mind in regard to prognosis.

Diagnosis.—The diagnosis of contracted kidney can be made with certainty only by examining the urine. We must, therefore, dwell again on the necessity of making this examination in all suspicious cases, because only in this way can we avoid overlooking the condition. The suspicion of a developing renal contraction should demand an examination of the urine, especially in all cases where the patient complains of frequent headache, of congestive conditions, of palpitation and dyspnœa, asthmatic attacks, disturbances of vision, general dullness, and dyspeptic symptoms, without finding any other reason for these symptoms. The polyuria, the clear urine of low specific gravity, containing a slight amount of albumen, in connection with the signs in the circulatory apparatus, the tense pulse, and the hypertrophy of the left ventricle, permit us to recognize the disease correctly in most cases. If retinal changes are present, they may sometimes be of much aid in confirming the diagnosis. The ætiological conditions—lead, gout, alcoholism, etc.—of course also merit attention.

The diagnosis presents great difficulty in the quite rare cases where albuminuria is absent. In these cases we are sometimes able to reach the correct interpretation of the morbid condition only by repeated examinations of the urine. Otherwise we can scarcely avoid mistaking it for chronic affections of the heart, such as myocarditis or idiopathic hypertrophy.

The diagnosis is also very difficult if the patient does not come under observation until the stage of fully developed disturbance of compensation. The characteristic features of the urine of contracted kidney are then absent, the urine is scantier, darker, richer in albumen, and it is often scarcely possible to decide whether we have a primary renal affection with secondary cardiac hypertrophy or a primary heart disease with a secondary congested kidney. If general arteriosclerosis or marked pulmonary emphysema is present at the same time, the judgment as to the condition is still more difficult. In such cases a correct diagnosis is possible only by very carefully balancing all the different symptoms, and considering the whole course of the disease.

Finally, the diagnosis of contracted kidney is very difficult in cases where the first examination of the patient is made during a sudden attack of uræmia or after an apoplectic seizure. Here the albuminuria is the symptom which

points most to the existence of a renal disease, although, in spite of this symptom, the judgment as to the condition, and its differentiation from other acute cerebral affections, often presents great difficulties.

We must devote a few words to those not very infrequent cases in which a small amount of albumen is found, often quite by chance, in the urine of youthful individuals who are apparently in perfect health; thereupon the question is propounded, which is often of extreme practical importance, whether we are dealing with a so-called physiological or intermittent albuminuria (*vide supra,* page 725), or with an actual renal disease—viz., an unsuspected contracted kidney. The diagnosis is never easy. In the first place, it is necessary to make careful observations for a considerable period, at the same time varying the conditions as to nourishment (nitrogenous diet, milk diet, use of beer) and activity (complete physical rest, persistent exercise); then we must carefully investigate all the possible ætiological circumstances, such as previous disease and mode of life, and finally take into consideration the associated phenomena—for instance, of the circulatory apparatus, the retina, etc. From a practical point of view it is advisable to be most cautious in all these cases, and, at any rate, to give such advice as to regimen with the aim of preventing damage to the kidneys, as if there were an actual renal disease.

Treatment.—As soon as the diagnosis of renal contraction is established, the whole hygienic condition of the patient must be regulated so as to prevent the advance of the affection in every possible way. In this respect two indications are to be fulfilled—to guard against any irritation which may have an injurious action on the kidneys and to relieve the work of the heart as much as possible, in order to keep off cardiac insufficiency as long as we can. The diet must be carefully regulated, and must be of scant measure or abundant and strengthening, according to the patient's physical constitution. In these cases, too, milk is a food of great value, but it should not be prescribed unnecessarily for corpulent patients. The determination as to the amount of salt in the food must depend on the considerations already pointed out. The use of meat is, in general, to be limited, while easily digestible dishes made from cereals and eggs, and vegetables and fruits, are to be recommended. Whether the so-called white meats (poultry, veal, lamb), as so many at present assume, are really less injurious than the so-called dark meats (beef, venison), cannot be positively proved, but, notwithstanding, this view will usually be remembered in practice. Alcoholic drinks should be permitted only in small quantity. Mineral waters (Fachingen, Wildungen, Wernazer water, etc.) and water with lemon juice or the juice of other fruits, etc., serve as beverages. Great care of the skin is an important matter. We should recommend the regular employment of warm baths or salt baths, and daily sponging of the whole body with brandy or warm vinegar. All physical overexertion is to be avoided, although moderate methodical exercise is to be recommended for corpulent patients. We should always provide for regular evacuation of the bowels by appropriate remedies, dietetic prescriptions, fruits, bitter mineral waters, etc. The general condition is often materially improved by proper air and recreation, and in this way resort to a bath may be of service, chosen according to the individual conditions, such as Brückenau, Marienbad, Carlsbad, Kissingen, Nauheim, Ems, or Baden-Baden. In ap-

propriate cases a winter residence in the south is advisable—e. g., in the Riviera or Egypt.

If disturbances of compensation appear, we must be still more strict in our dietary, and must enjoin the utmost physical rest, at the same time employing other treatment according to the symptoms which may be present. Above all, we must endeavor to impart new vigor to the heart by means of digitalis and digitalis preparations—digalen, digipuratum—though unfortunately such efforts often prove unavailing. The minutiæ of treatment in this connection are almost precisely the same as in chronic heart disease (*q. v.*) and in other renal diseases. The treatment of the dyspnœa and chronic uræmia, which develop in the last stages of the disease, is apt to be most unsatisfactory; but we can, sometimes, at least, remove the symptoms by various external applications upon the chest and the head (ice bags or hot compresses), by various drugs to combat the nervous symptoms, bromid of potassium, antipyrin, nitroglycerin, and by the combination of cardiac stimulants, such as digitalis and strophanthus, with diuretics, such as caffein, diuretin, agurin, theophyllin, theocin, etc. In the worst cases narcotics are indispensable, particularly subcutaneous injections of morphin. As yet we are unable to exert a direct beneficial influence by drugs upon the interstitial process in the kidneys. The only ones recommended for this purpose and worth a trial are the preparations of iodin—viz., iodid of potassium or iodid of sodium, in daily amounts of 3 to 8 gr. (gm. 0.2 to 0.5), and, in anæmic patients, iodid of iron. We do not need to go minutely into the treatment of the numerous complications which occur.

The prophylaxis of renal contraction is evident; we should avoid so far as we can the known ætiological conditions.

CHAPTER V

AMYLOID KIDNEY

Ætiology.—The amyloid kidney is invariably associated with the more or less extensive amyloid degeneration of other organs in the body. In its clinical relations, however, it claims the most interest of all amyloid diseases, since it has by far the greatest significance for the whole clinical picture of amyloid degeneration.

As is well known, we understand by amyloid degeneration a peculiar change which, under certain pathological conditions, is observed in the connective tissue, and especially in the smaller vessels. The walls of the vessels are thickened, they have a lustrous, homogeneous appearance, and they show peculiar reactions on treatment with certain coloring agents. These reactions are due to the presence of an albuminoid substance—amyloid—which either is deposited in the tissue from the blood, or, as is much more probable, is developed in that spot from the albuminoid substances there present. In marked amyloid degeneration the diseased organs often show macroscopically an altered, "bacon-like" appearance, and assume a characteristic red-brown color on treating the affected parts with Lugol's solution of iodin, changing

to violet on the addition of sulphuric acid. The microscopic examination alone affords more accurate conclusions as to the presence and distribution of the degeneration. For this purpose we usually stain the tissues with methyl violet or gentian violet. The amyloid portions thus take on a very characteristic and clearly defined red color. In this way we can discover that the amyloid degeneration begins everywhere in the walls of the small vessels, that the interparenchymatous connective tissue may also be affected later, but that the parenchymatous cells proper, liver cells, renal epithelium, etc., almost always remain perfectly free. The latter often show atrophic and fatty degenerative changes (*vide infra*), but little if any amyloid degeneration.

Nothing is known as to the special causes which effect this peculiar metamorphosis of the albumen of the connective substance into amyloid. We know only that there are a number of primary diseases in which experience has shown that amyloid degeneration quite frequently develops as a secondary condition in the different organs. These conditions have, for the most part, the common characteristic of being associated with general cachexia and debility, and of being connected with some localized chronic morbid process in some part of the body, from which abnormal chemical matters may be constantly carried into the blood. We suppose that the normal albuminous material is transformed into amyloid by means of such influences. With regard to the relations which may exist between the so-called hyaline degeneration of Recklinghausen, and amyloid disease, the investigations are not yet complete.

The conditions in which amyloid degeneration in general, and consequently amyloid disease of the kidney also, are chiefly observed, are the following, arranged in about the order of their frequency:

1. Chronic pulmonary tuberculosis, particularly the ordinary ulcerative phthisis. Tubercular ulcers of the intestines, with or without coexisting marked pulmonary tuberculosis, may also lead to amyloid disease.

2. Long-continued chronic suppuration in the bones or soft parts, especially chronic fungous processes with fistulæ into the bones or joints, empyema with fistulæ, vertebral caries, etc.

3. Constitutional syphilis, chiefly the cases with ulcerative and usually tertiary processes in the bones and mucous membranes.

4. Other ulcerative processes, or processes associated with chronic suppuration: saccular bronchiectases, chronic intestinal ulcers (for example, of dysenteric origin), purulent pyelocystitis, vesicovaginal fistulæ, ulcerated new growths, such as cancer, etc.

5. In rare cases amyloid degeneration has also been observed in other chronic diseases, as in malaria, gout, and other chronic articular affections. In the medical clinic at Leipsic we once saw a case of marked amyloid kidney in a girl of twenty-one with aortic insufficiency.

6. Finally, in a small class of cases, of which we have ourselves seen some examples, no discoverable cause at all may be found at the autopsy for quite extensive amyloid degeneration. In such cases, therefore, there seems to be a primary intoxication with resultant amyloid disease.

Pathological Anatomy.—With regard to the anatomical lesions of amyloid disease in other organs, we shall refer to handbooks on pathological anatomy.

As to amyloid liver, see page 714. Here we shall discuss merely the pathological anatomy of amyloid kidney.

In very slight and limited amyloid degenèration of the kidneys, the latter present a perfectly normal appearance to the naked eye. Careful microscopic examination alone shows amyloid degeneration of the walls of different vessels in the cortex, and especially in the medullary substance.

The commonest and most characteristic form of amyloid kidney is the so-called large white amyloid kidney (waxy kidney, lardaceous kidney). The kidney is usually enlarged, and the surface is smooth and of a grayish-white or yellowish color, and usually somewhat mottled. On section, the cortex is wide and also of a yellowish-white color, and the glomeruli may often be recognized with the naked eye as dull, lustrous, translucent points. Hemorrhages are scarcely ever seen. The medullary substance may be also pale, or darker. In many cases the cortex may itself have a darker reddish or mottled appearance, which is due merely to the greater amount of blood in the organ. The pale-yellow color is due either to the anæmia or to the fatty degeneration, while the amyloid spots show a more translucent character with a bacon-like luster.

If we examine the kidney microscopically, we find first the amyloid degeneration, which, in varying extent and combination, affects most frequently the glomeruli and also the capillaries of the cortex, the vasa recta, and sometimes the membranæ propriæ of the uriniferous tubules. In pure amyloid kidney the rest of the renal tissue is normal, but in many cases we find changes in the epithelium—fatty degeneration, desquamation, and disintegration—and also not infrequently interstitial cellular infiltration. We see, therefore, that amyloid degeneration of the kidney is not infrequently associated with degenerative changes of an inflammatory nature. Thus we observe, not infrequently, the combination of chronic diffuse nephritis (large white kidney) with amyloid.

If the process has lasted for a long time, it leads, as in ordinary nephritis, to a complete atrophy of tissue in some parts, with a corresponding increase of connective tissue. Then the renal tissue sinks in at the affected spots, and there is a decided unevenness to the surface of the kidney. There is even a completely developed red or white contracted kidney, in which we find abundant amyloid, and which is, therefore, termed amyloid contracted kidney. In this form the parenchymatous and interstitial changes correspond precisely to those in ordinary contracted kidney, only the amyloid degeneration is added to them.

At present differences of opinion prevail as to the precise connection between amyloid and the inflammatory degenerative processes in the kidney. In our opinion there is, in most cases, an actual combination of both conditions, they being coeffects of simultaneously acting causes. We have seen that in tuberculosis, chronic suppuration, etc., genuine nephritis may develop, as well as amyloid disease, so it cannot be surprising that sometimes with these diseases both sequelæ, nephritis and amyloid, should develop side by side, and that we should, consequently, find in the kidney not only the changes associated with an inflammatory, large, white kidney, a secondary contraction, or a genuine contracted kidney, but also a more or less extensive amyloid de-

generation. On the other hand, it, of course, cannot be questioned that the disturbance of circulation, which must arise in consequence of a marked amyloid degeneration of the vessels, is of influence on the nutrition of the renal tissue, and that, therefore, many changes in it, especially fatty degeneration of the epithelium, are, under some circumstances, the direct result of the amyloid disease.

Clinical History.—If we consider the great diversity which the distribution of the amyloid change in the kidneys shows, and its manifold combinations with inflammatory processes, it is clear from the outset that we cannot get up a uniform picture of the symptoms of amyloid disease in general. To this we must add that the symptoms of amyloid disease, which is almost always a secondary condition, are also modified in various ways by the primary disease.

We must first state that many cases, where the amyloid change in the kidneys is of comparatively slight extent, cannot be recognized by any clinical symptom. The albuminuria in particular may be entirely absent, as has been repeatedly proved (Rosenstein, Litten, and others). That in such cases the vasa recta and not the glomeruli are chiefly affected by the amyloid degeneration has been affirmed but not proved.

Except in these instances, however, the urine secreted from the amyloid kidneys shows marked changes, which, of course, present considerable variations according to the form of the individual case. The amount of urine is most frequently about normal, or somewhat diminished—in some cases much diminished—but in others it is decidedly increased, so that the patient may pass 80 to 120 ounces (2,500 to 3,500 c.c.) in the twenty-four hours. We quite frequently see considerable variations in the amount of the urine in the same patient at different times. All these differences are easily explained if we remember how many circumstances may affect the amount of urine—the presence or absence of inflammatory changes in the kidney, the presence or absence of cardiac hypertrophy, coexisting perspiration, diarrhea, œdema, fever, etc.

The color of the urine is almost always light yellow. Only exceptionally, in amyloid nephritis, does it contain an abundant sediment; usually it is entirely, or almost entirely, clear. The very considerable amount of albumen in the urine, which is often one or two per cent, is also characteristic of amyloid kidney. In many cases, indeed, particularly in the combination of amyloid with contracted kidney, the amount of albumen is smaller, although usually decidedly more abundant than in pure interstitial nephritis. Senator has pointed out that the comparative amount of the globulin which is contained in the urine besides the serum albumen is often particularly large in amyloid kidney. The specific gravity of the urine varies very much according to the amount of water and albumen in it. It may be increased (1.015 to 1.020) or diminished (1.010 to 1.005).

If we examine the urine under the microscope, we usually find only a few hyaline casts, and also most frequently a small number of white blood corpuscles. In general, it is very characteristic of amyloid kidney that there is an abundance of albumen, contrasting with the scanty or scarcely appreciable microscopic sediment. In the combination of amyloid with more marked nephritic changes the sediment is more abundant, so that the urine is cloudy.

The microscope then shows more numerous hyaline or moderately fatty casts, more abundant white blood corpuscles, sometimes a little renal epithelium, and in quite rare cases even red blood corpuscles. Amyloid reaction is said to occur in the casts, but it is very rare, and therefore of no value in diagnosis.

The other morbid symptoms which are observed in amyloid kidney depend either upon the change in the kidneys themselves, or upon coexisting amyloid degeneration in other organs; or, lastly, upon the primary disease. The symptoms of the latter are, of course, extremely varied, but in many cases they may be entirely subordinate.

In regard to the directly resulting symptoms of amyloid kidney, their occurrence is of interest, especially in comparison with the analogous conditions in acute nephritis. Dropsy of a moderate, or even a severe degree, is often present in amyloid kidney, but it may also be entirely absent. We must remember that an œdema independent of a renal affection may be produced by marantic venous thrombosis. Uræmic symptoms are distinctly rare in amyloid kidney, but they are sometimes observed, especially in their milder forms, such as vomiting. It is a very important point that hypertrophy of the left ventricle is absent in most cases of amyloid kidney. This is explained partly by the fact that the disease usually affects feeble, cachectic individuals who have no superfluous material for the manufacture of cardiac hypertrophy, and partly by the fact that in pure amyloid kidney there is no appreciable retention of urinary constituents in the blood. This latter fact explains, also, the infrequency of uræmic symptoms (*vide supra*). If, however, the amyloid degeneration is combined with genuine nephritic changes, the condition of things is altered. In amyloid contracted kidney we have repeatedly observed hypertrophy of the left ventricle.

Albuminuric retinitis hardly ever appears in pure amyloid kidney. In the amyloid contracted kidney it has sometimes been observed, however, in cases where there has probably been originally a pure contracted kidney, with amyloid coming on later. The secondary inflammations in the internal organs, such as renal pneumonia, and the hemorrhages, such as cerebral hemorrhage, are also rare.

The patient's general condition is dependent in part upon the renal disease, and in part upon amyloid degeneration in other organs, but mainly upon the primary disease. The patient with amyloid kidney is usually, therefore, cachectic, and shows in high degree a pallid, anæmic color of the skin. If, also, general dropsy develops, we have an external appearance very characteristic of amyloid disease; still, in some few cases of syphilis, bronchiectasis, and unilateral pulmonary contracture, the general nutrition remains tolerably good for a considerable time.

The symptoms which point to a coexisting amyloid degeneration in other organs besides the kidneys are of great diagnostic significance. The symptoms in the liver (enlargement, abnormal firmness, and a hard, sharp lower edge to the organ), the spleen (enlargement and hardness), and intestines (obstinate diarrhea not yielding to any remedy) are clinically important in this respect. The interpretation of the diarrhea is, of course, usually difficult, since it may often depend upon tubercular intestinal ulcers as well as upon amyloid disease of the intestines.

We can scarcely make general statements in regard to the whole course and the duration of amyloid kidney, since the form of the primary disease is to be especially considered in these cases. In regard to the time that it takes for an amyloid degeneration to develop in an existing primary disease, the degeneration is certainly present sometimes after a few months. Of course, it is hardly ever possible to determine its onset accurately, since the first beginnings of amyloid degeneration in the kidneys do not usually permit themselves to be recognized at once by the appearance of albuminuria (*vide supra*). The duration of amyloid kidney varies very much according to the severity of the case; it may last only a few weeks or months before death, while other cases have certainly lasted for a year, especially in amyloid contracted kidney.

Prognosis.—The prognosis of amyloid kidney is in most cases utterly unfavorable, which is due mainly to the incurability of the primary disease; but trustworthy observers have repeatedly proved that, when the primary disease is curable, as with syphilis and many chronic suppurations, an already developed amyloid kidney may exceptionally be completely restored.

Diagnosis.—The diagnosis of amyloid kidney can be made with considerable certainty when the evident signs of a renal affection are added to those of a disease which we know by experience to promote the development of amyloid degeneration. Whether in such cases we have a pure amyloid or a pure nephritis, or a combination of the two, can be decided with some certainty only from the condition of the urine; a clear urine, containing but few morphological elements, but rich in albumen, points to pure amyloid, while a large number of casts and red and white blood corpuscles in the urine points to the presence of inflammatory changes in the kidney. The diagnosis of amyloid contracted kidney is justified when the symptoms of contracted kidney (abundance of pale urine, secondary cardiac hypertrophy) are seen in association with such morbid conditions as are likely to occasion amyloid disease. Of great importance, as we have already pointed out, is the comparatively large amount of albumen and the consequently somewhat higher specific gravity of the urine. Of many cases of amyloid kidney it is very characteristic, and so of diagnostic value, that there is a rapid and frequent variation in the amount of urine and the percentage of albumen (Wagner).

A very material support for the diagnosis of amyloid kidney, and therefore one which should always be looked for, is the discovery of amyloid degeneration in other organs. We have briefly mentioned above the most important symptoms in the liver, the spleen, and the intestines referable to this point.

Treatment.—Only the treatment of the primary disease can, of course, be considered, both as a prophylactic and also as a causal indication. In many surgical cases, and also in the cases of amyloid in syphilis, there is a possibility of this (as by iodid of potassium), but otherwise we try to improve the primary disease as far as we can.

In other respects the treatment is purely hygienic and symptomatic. We must try to strengthen the patient as much as possible by good food and the exhibition of preparations of iron and quinin. The use of iodid of iron or small doses of iodid of sodium is to be recommended. In a symptomatic point of view the same remedies are used as in other renal diseases.

We insert here a synopsis of the condition of the urine and of the left ventricle in the most important varieties of renal disease.

	Amount of Urine.	Specific Gravity.	Amount of Albumen.	Morphological Constituents.	Condition of Left Ventricle.
1. Acute nephritis...	Diminished.	High.	Considerable.	Numerous casts; blood corpuscles; epithelium.	Not hypertrophied.
2. Chronic diffuse nephritis.....	Normal or somewhat diminished	High.	Considerable.	Numerous casts; blood corpuscles; epithelium; fatty degeneration of the cells.	Hypertrophied.
3. Contracted kidney.....	Increased.	Low.	Slight.	Few casts; small number of blood corpuscles.	Hypertrophied.
4. Amyloid kidney..	Variable, normal, or increased.	Variable.	Considerable.	Few casts.	Not hypertrophied.
5. Amyloid contracted kidney .	Increased.	Rather low.	Moderate.	Few casts.	Hypertrophied.
6. Passive congestion.....	Diminished.	High.	Absent or scanty.	Few casts; blood corpuscles.	Generally primary heart disease.

CHAPTER VI

PURULENT NEPHRITIS AND PERINEPHRITIS

1. PURULENT NEPHRITIS

Ætiology.—Although in the forms of nephritis so far described the occurrence of large numbers of nuclei in the interstitial tissue has been repeatedly mentioned, none of them ever come to genuine suppuration —that is, to a purulent liquefaction of tissue, a true abscess formation. The origin of a purulent nephritis is, rather, always associated with the entrance of perfectly definite morbid irritants into the kidneys. These are invariably organized, and their special peculiarity is to excite a purulent inflammation.

There are two chief ways through which the morbid irritants may reach the kidneys—the arterial blood current and the urinary passages. The first-mentioned means of entrance is to be considered in all the cases of purulent nephritis which come on as one symptom of pyæmic processes and certain forms of ulcerative endocarditis (see pages 138 and 373 on the point). Far more rarely purulent nephritis develops in this way as a complication in other diseases, such as dysentery. Purulent nephritis also occurs in actinomycosis (Israel).

The excitants of inflammation take the second path in those cases where a purulent nephritis follows an inflammation of the lower urinary passages, the

pelvis of the kidney, the bladder, etc. Here the bacteria, which almost always enter directly into the urinary passages (the urethra and bladder) from without, pass gradually upward from the bladder through the ureter to the pelvis of the kidney; from this they enter the apertures of the collecting tubes and the uriniferous tubules of the kidney, everywhere exciting a purulent inflammation. We therefore term these forms of purulent nephritis—with regard to their origin—purulent pyelonephritis.

We must remark in conclusion that a purulent nephritis and perinephritis (*vide infra*) may arise in direct wounds of the kidney from infection of the wound.

Pathology.—Purulent nephritis shows quite characteristic peculiarities and differences according to its mode of origin. (We omit traumatic abscesses here.)

The renal abscesses in pyæmia and analogous diseases are usually focal suppurations, which only exceptionally attain a great extent, but which are usually to be recognized with the naked eye as numerous little yellowish dots or lines, scattered over the whole kidney, about half a millimeter or a millimeter in diameter. On microscopic examination, these nodules prove to be genuine little abscesses, in whose territory the renal tissue proper is completely destroyed. In the middle of them we often find the originating colony of micrococci, the "micrococci embolus," seated in a central vessel. The conditions are still plainer if we examine an earlier stage of the process. We find vessels (the loops of the glomeruli, or the encircling capillaries), which are completely plugged with micrococci, and in whose vicinity the renal tissue is still perfectly normal. We further see analogous spots where the renal tissue is already necrosed in the vicinity of the colony of micrococci, and is infiltrated with emigrated cells. These nodules show, finally, a continuous transition to the completed abscess, which is often surrounded by a hyperæmic or even a hemorrhagic areola.

In purulent pyelonephritis the renal abscesses appear somewhat different. The abscesses also have a characteristic striated appearance, corresponding to the distribution of the inflammation along the straight tubules. They often extend from the point of the renal papilla through the cortex to the surface of the organ, so that from the outside we see the abscesses, showing through as yellowish points. The broader abscesses arise from the confluence of neighboring striæ. The microscope shows that the purulent inflammation arises from the vessels of the interstitial tissue, in whose territory the uriniferous tubules are, of course, destroyed. The clusters of micrococci form the most interesting feature. These settle originally in the uriniferous tubules, and are the special cause of the necrosis of epithelium and the inflammation. Pyelonephritis, indeed, was one of the first diseases for which a bacterial origin was discovered (Klebs).

Clinical Symptoms.—We can speak very briefly here in regard to the clinical symptoms of purulent nephritis, since they can never be sharply separated from the symptoms of the primary disease. The pyæmic renal abscesses, and the abscesses in ulcerative endocarditis, hardly ever cause special clinical symptoms, so that their presence is first recognized on the autopsy table. Since the abscesses do not, as a rule, communicate with the uriniferous tubules, there is usually no great amount of pus in the urine.

The clinical symptoms of pyelonephritis also depend less upon the nephritic abscesses than upon the previous and accompanying pyelitis and cystitis. We will therefore return to renal abscesses in the description of these diseases.

2. PERINEPHRITIC (PARANEPHRITIC) ABSCESS

Perinephritic abscess is the name given to suppurations in the vicinity of the kidney, especially in its fatty capsule or in the perirenal connective tissue. Apart from any traumatic origin for such abscesses, they develop most frequently as a result of purulent nephritis or purulent pyelitis. The escape of pus, which involves the surrounding tissue in the inflammation, may come from the ureter or pelvis of the kidney, or from the kidney. The special form of primary disease differs very much; it may be either simple purulent pyelitis, or pyelitis from renal calculi, or sometimes tubercular processes and new growths that finally suppurate, such as cancer or echinococci. The perirenal suppuration may also take its start from the other organs in the neighborhood. Thus cases have been seen in which the perinephritis followed a perityphlitic abscess, a hepatic abscess, or a psoas abscess after vertebral disease. Perinephritic suppuration may also be due to actinomycosis. It is a very important fact, however, that paranephritic abscesses may develop as a primary disease in persons in apparently perfect health, particularly in middle-aged men, and this without any discoverable cause. It is usually absolutely impossible to determine how and by what path the inflammatory germs reach the perirenal connective tissue unless possibly they migrate from the intestine. These cases of abscess are very important from a clinical standpoint, because the symptoms are at first difficult of interpretation. There is fever, usually of a decidedly intermitting pyæmic type, and there are also dull, indefinite pains in the loins or abdomen, constipation, and constitutional symptoms. The cause of all these symptoms is gradually made apparent by the development of distinct local changes.

In almost all cases of perinephritic abscess the accumulation of pus finally becomes so great that a swelling appears, usually in the lumbar region, and this grows more and more prominent; at first it is scarcely noticeable, but later the skin becomes œdematous there, it constantly protrudes more and more, it assumes an inflammatory hyperæmic redness until, finally, a definite fluctuation shows the advance of the abscess up to the skin. In other cases the inflammatory swelling extends forward into the iliac fossa; then there is also abnormal resistance and dullness above Poupart's ligament. The swelling may also extend upward toward the diaphragm, so that the diaphragm is crowded upward, giving rise to marked dyspnœa. The relations of the swelling to the descending colon are sometimes the same as in new growths of the kidney (compare Chapter VIII).

Besides the swelling there is almost invariably a very great pain in the affected region, either spontaneous or on pressure. If the swelling presses on the large nerve trunks in the vicinity, it produces severe shooting pains in the leg of the same side, and sometimes a numb feeling and paresis. The leg is then often kept in a position similar to that in coxitis.

The fever is persistent, and usually of an intermittent character and associated with rigors. It and the pain make the patient thin and feeble, even

to an extreme degree. The urine does not contain pus unless the abscess has some connection with the urinary passages.

Recovery may occur if the pus finds some way out of the body. Apart from operative interference, the most favorable termination is the spontaneous discharge of the pus into the intestine (colon), or into the urinary tract (pelvis of the kidney, bladder). The course of the disease is much more tedious if the pus finds its way through the skin. If renal abscesses discharge outwardly, they most frequently point in the loins, less often like a psoas abscess, below Poupart's ligament. They may also discharge into the pleural or peritoneal cavities, with a consequent empyema or quickly fatal peritonitis. In other cases, if prompt surgical aid is impossible, death occurs from exhaustion.

Diagnosis.—The diagnosis is made chiefly from the swelling, pain, and fever, and a consideration of the ætiological factors. We can settle any doubts in most cases by exploratory puncture. The condition may be confounded with hydronephrosis, psoas abscess, and solid tumors of the kidney.

Treatment.—The only treatment, apart from the fulfillment of any symptomatic indications, is surgical, and consists in opening and draining the abscess. The result depends chiefly upon the patient's general condition, and the character of the primary disease. The details are to be found in the text-books of surgery.

CHAPTER VII

DISTURBANCES OF CIRCULATION IN THE KIDNEYS

1. The Congested Kidney.—Although local impediments to the flow of venous blood from the kidneys, such as thrombosis of the renal veins, hardly ever attain a clinical significance, the participation of the kidneys in a general venous stasis, as is seen chiefly in heart disease (compare page 403), pulmonary emphysema, etc., is of great diagnostic importance, since we possess in the condition of the urine quite an accurate measure of the intensity as well as of the increase and decrease of the stasis.

The congested kidney is easily recognized anatomically. The organ is often somewhat enlarged, it feels firmer than normal, and shows, both on its surface and on section, a dark, bluish-red color—"cyanotic induration." The medullary substance is usually darker than the cortex. Under the microscope we see considerable dilatation and a tense fullness of the veins and capillaries. The parenchyma is normal, but in more advanced cases it may show a beginning fatty degeneration of the epithelium, which is a result of the defective arterial blood supply. At first the interstitial tissue is little changed, but if the congestion persists for a long while there may be a gradual destruction of the renal parenchyma, to a certain degree, with the formation of an abundance of contractile interstitial tissue (congested and contracted kidney).

The clinical symptoms of congested kidney concern only the changes in the urine. The amount of urine diminishes, corresponding to the diminution of the arterial pressure and the diminished rapidity of the blood current. Only 25 to 20 ounces (800 to 500 c.c.), or less, 10 to 6 ounces (300 to 200 c.c.), are secreted daily. The urine is also more concentrated and darker than normal,

and often has an abundant sediment of urates. If nutritive disturbances have begun in the epithelium of the glomeruli as a result of stasis, the urine is also albuminous, but the amount of albumen in pure congested kidney is always slight—about $\frac{1}{10}$ to $\frac{1}{6}$ of the volume. The urine often contains, besides, a few hyaline casts, and a few white and red blood corpuscles, the latter pointing to little congestive hemorrhages.

If the changes mentioned come on as one symptom of a general venous stasis, and are, accordingly, associated with cyanosis and dropsy, the diagnosis of congested kidney can be made with certainty. If we succeed in restoring the circulation by appropriate remedies, such as digitalis, the urine at once becomes more abundant and clearer and its albumen disappears. Otherwise the characteristics of the urine of passive congestion last until the patient's death.

2. **Embolic Infarction in the Kidneys.**—Since the renal infarction, although it has great pathological interest, is hardly ever of clinical significance, we will limit ourselves here to a brief description of the most essential points.

If one of the smaller renal arteries is plugged by an embolus in heart disease, the affected portion of the organ cut off from the circulation must perish, since all the renal arteries are terminal arteries. The epithelium undergoes the well-known changes of coagulation necrosis, disappearance of the nuclei of the cells, and disintegration, and the tissue becomes entirely or in part a hemorrhagic infarction (compare page 403). In this way arise the characteristic wedge-shaped, red, hemorrhagic infarctions in the kidney, or far more frequently the yellowish-gray, anæmic infarctions (often surrounded by a hemorrhagic areola), the base of which is at the surface of the kidney; the base may reach a width of half a centimeter to a centimeter or more, while the apex extends a varying distance into the cortex, or even into the medullary substance. Later on the gradually disintegrated tissue of the infarction is absorbed, round cells emigrate from without into the region destroyed, and a shrunken connective-tissue cicatrix gradually develops in place of the former infarction. Many kidneys may have such a granular surface from numerous infarction cicatrices that they may be appropriately termed "embolic contracted kidneys."

The anatomical processes just briefly described cause in most cases no special clinical symptoms at all. Sometimes, however, a slight amount of blood in the urine seems to depend on the development of a hemorrhagic infarction in the kidneys, so that when a cause for embolic processes, such as heart disease, is present, we may entertain the suspicion of the development of a renal infarction during life. In rare cases the development of a rather large infarction of the kidneys is associated with a sudden and violent pain in that region, with marked subsequent hæmaturia or, as we observed in one case, hemoglobinuria.

The embolic processes in the kidney never demand special treatment.

CHAPTER VIII

NEW GROWTHS IN THE KIDNEYS

1. Congenital Sarcomatous Mixed Growths of the Kidney.—Of the primary forms of tumor occurring in the kidney, those tumors, often of great size, that are not rarely observed in young children especially claim our interest. In former times, they were often regarded either as sarcomata or as carcinomata. We now know that we have to deal with new growths which owe their origin to embryonic disturbances of development, and which, therefore, present a varied formation even in one and the same tumor, being at one time more of a sarcomatous character and composed of spindle or round cells, at another more adenomatous or carcinomatous. It is an especially interesting fact that in these tumors tissue elements may appear which are normally never present in the kidney, and whose presence can be explained only on the theory of "scattered germs." Thus, striped muscle fibers have been repeatedly found in tumors of that character, to which the name striocellular myosarcoma or rhabdomyoma is applied; in other cases cartilaginous tissue, mucoid tissue, etc., have also been found. This theory obtains a further interesting confirmation from our own observation of the development of left-sided, and, probably, congenital renal sarcoma in two brothers. Both children died when between two and three years of age, and the autopsy gave almost precisely the same lesions in both: numerous metastases in the liver and lungs, besides a new growth almost as large as a child's head in place of the left kidney. But these tumors may also attain a very considerable size without forming metastases.

2. **Renal Sarcoma and Carcinoma.**—Genuine renal sarcoma and particularly carcinoma are also not rarely observed. The carcinoma may originate in part from the tissue itself, and, in part, from the membranous lining of the pelvis of the kidneys without giving rise to metastases. The sarcomata are, in part, angiosarcomata and endotheliomata, etc. Renal cancer is also remarkably frequent, comparatively speaking, in children under four years of age, and about equally common in the two sexes. Of course, we find renal cancer in persons of more advanced years; in some cases of this sort renal calculi seem to occasion the development of carcinoma. This is like the relation between gallstones and cancer of the biliary passages. Usually only one kidney is affected, chiefly the left, as it seems, but the new growth has sometimes been found in both kidneys. In its character, renal cancer belongs either to the denser or to the softer, medullary form. It may permeate the whole kidney and change it to a large tumor, weighing 15 or 20 pounds (5 or 10 kgm.). Softening and hemorrhage very often take place within the tumor. The proliferation has been repeatedly observed to extend to the neighboring parts, especially the pelvis of the kidney, and metastases also form in other organs, as in the lymph-glands, liver, or lungs. It should also be mentioned that several times renal cancer and cancer of the testicle have been combined.

3. **Benign Kidney Tumor.**—The only benign form of kidney tumor that has any particular clinical interest is the rare one of lipoma, since it may develop into a large-sized tumor. Besides this, we would mention the interesting cystic tumors of the kidneys (cystic degeneration), which probably have

some connection with anomalies of embryonic development. Their occurrence as a congenital anomaly, their occasional appearance as a family disease, and, finally, their frequent association with other disturbances of development (anomalies of the ureters and of the renal vessels, simultaneous formation of cysts in the liver, etc.), all point to this conclusion. Some kidney cysts, which do not proceed from the uriniferous tubules, but from atypical proliferations of glands, are to be regarded as adenocystomata. Kidney cysts may develop into rather large tumors, and may be unilateral or bilateral.

4. **Hypernephroma.**—Clinically, the so-called hypernephroma—i. e., growths originating in scattered suprarenal cells, and therefore lying beneath the kidney capsule ("Grawitz tumors")—are not to be separated from genuine tumors of the kidney. They may form large tumors whose histological structure allows their origin from the suprarenal cells to be plainly recognized. In consequence of the large amount of fat in them, these swellings generally present a yellowish-white soft surface on section, and a tendency to disintegration and to hemorrhages.

Symptomatology.—The clinical symptoms of new growths in the kidneys may first become noticeable through the appearance of a swelling. Tumors of the kidney, in children, may attain a considerable size (*vide supra*), causing a distention of the entire abdomen. But even when smaller, upon careful palpation, renal tumors often enough are distinctly felt in the lumbar and lower lateral abdominal regions. Bimanual palpation is most advisable, the one hand pressing forward from behind and below in the region of the kidney, while the other presses against it from above and in front. In this manner it is often possible to get the entire tumor between the two hands. A particularly characteristic factor in diagnosis is the *ballottement rénal* described by Guyon: when a gentle thrust is made in the renal region the tumor is felt to strike softly upon the anterior abdominal wall. The tumor feels firm and sometimes smooth, sometimes uneven; it does not usually move with respiration, but this rule has exceptions, particularly in the case of tumors of the right kidney. In tumors of the left kidney some diagnostic importance attaches to the relation of the new growth to the descending colon, inasmuch as the latter is pushed forward by the growth of the tumor and comes to lie between it and the anterior abdominal wall. It is quite often possible to demonstrate that portion of the intestine which traverses the tumor by means of percussion at times after previous distention with air, and sometimes also by means of palpation, or by Röntgen rays after the introduction of bismuth pap. In the case of tumors of the right kidney there may be corresponding relations to the ascending colon, but this is less frequent. Almost all larger tumors of the kidney occasion displacement of the diaphragm upward, and of neighboring organs laterally. A number of other clinical symptoms are to be noted. These sometimes become noticeable before the appearance of a palpable tumor, and themselves lead to a more exact palpatory examination of the abdomen. In a third class of cases it is altogether impossible directly to demonstrate the existence of the tumor, and only the accompanying and resultant phenomena appear. There may often be scarcely any tenderness and pain, but in some cases the latter is violent and persistent. Often the pressure of the tumor upon neighboring nerve trunks—for instance, the sciatic—occasions obstinate neuralgia, sometimes associated with paresis.

The state of the urine is of the greatest importance. Not infrequently, it shows no abnormal conditions at all if it is secreted by the other healthy kidney, or by the normal parts of the diseased one. Very often, however, from time to time, blood appears in greater or lesser amount mixed with the urine. In many cases the hæmaturia is the first indication of an existing kidney affection. It is, of course, rare in congenital tumors in children, but, on the other hand, quite frequent in sarcoma and, above all, in cancer in adults, and equally frequent in hypernephroma. As has already been mentioned, it often appears at a time when no tumor at all can be felt. The hemorrhage is associated with colicky pains only when large clots have to pass through the urinary passages. Sometimes, but very rarely, small particles and shreds of tissue from the disintegrated new growth may be found in the urine.

The influence of the tumor on the constitutional condition varies greatly. The general condition of children with congenital tumors, may remain good for a long time. In other respects the symptoms are the same as in all other tumors. The appearance of mild symptoms of Addison's disease repeatedly observed by us in hypernephroma, is very interesting. We should, therefore, carefully note any anomalies of pigmentation in the folds of the hands and fingers, etc. We have also seen peculiar nervous conditions (persistent somnolence, comatose state) in such cases. We must also mention the peculiar symptom several times observed in children with congenital renal tumors—viz., an abnormally early development of the pubic and axillary hairs (Kühn). The tendency of many renal tumors to form metastases in the glands, the lungs, the brain, etc., is clinically important. I have repeatedly seen cases of "brain-tumor," which, at the autopsy, turned out to be metastases of small primary renal tumors. In conclusion, we would mention that there are cases of bilateral cystic kidneys whose clinical picture resembles that presented by contracted kidney, viz., excretion of a pale urine with only a small amount of albumen, development of cardiac hypertrophy, and finally uræmia.

Diagnosis.—The diagnosis of renal tumors is, in many cases, quite evident, but in others very difficult. The position of the new growth, its limited mobility, its relations to the colon, the possibility of grasping and moving the tumor between the hands on bimanual palpation, one hand being behind the kidney and one in front, and, above all, our knowledge as to the occurrence of renal tumors in children, often suggest the correct interpretation of the case. In older persons renal hemorrhages which cannot be otherwise explained must direct our suspicions to the possibility of a cancer of the kidney. Often, however, the disease has been confounded with tumors of the retroperitoneal glands and of the ovaries, with large psoas abscess, tumor of the liver, and splenic tumor. We can only briefly call attention to the importance of a cystoscopic examination.

Prognosis.—The prognosis is, of course, unfavorable. The disease sometimes lasts only a few months, sometimes a year or two, rarely longer.

Treatment.—The treatment must in most cases be purely symptomatic. The only expectation of success lies in the operative removal of the new growth, the details of which are to be found in recent monographs on renal surgery.

CHAPTER IX

PARASITES OF THE KIDNEYS AND OF THE URINARY PASSAGES—CHYLURIA

1. Echinococcus of the Kidney.[1]—Echinococcus cysts have been repeatedly found in the kidney, although much more rarely than in the liver. Usually only one kidney is affected, and the parasite is generally situated in the renal substance itself, only exceptionally between it and the capsule of the kidney. The size of the echinococcus cysts may be very considerable, the diameter reaching to 20 cm. or more.

Clinical *symptoms* usually first appear when the tumor can be felt through the abdominal walls. Subjective symptoms may even then be entirely wanting. Pain on pressure develops gradually later. The tumor usually has an approximately globular shape. Its relations to the neighboring organs, especially to the colon, are the same as we have learned to recognize in the preceding chapter, in the description of cancer of the kidney. The so-called hydatid thrill which is said to be characteristic, and which is obtained by giving the tumor a little push with the flat of the hand, can be distinctly felt in only the rarest instances.

It is a comparatively frequent occurrence for the echinococcus cyst to burst into the pelvis of the kidney. Then single echinococcus cysts, or at least bits of membrane, hooklets, etc., are passed with the urine, usually with severe colicky pains, which are exactly like the renal colic from the passage of a calculus. Such attacks may be often repeated, and may form a very severe type of disease by obstructing the urinary passages—the bladder and urethra. In such cases the symptoms of a secondary pyelitis and cystitis are often added.

Perforations in other directions are much rarer. The rupture of a renal echinococcus into the lungs has sometimes been observed, the patient coughing up echinococcus cysts.

Sometimes, especially after injuries, the sac of the echinococcus becomes inflamed, suppurates, and leads to a general pyæmic condition.

The diagnosis of renal echinococcus is possible only when a tumor can be made out belonging to the kidney, and when portions of echinococcus are passed with the urine, or through an exploratory puncture. It has already been stated that one should be cautious about employing this procedure (see page 710). The cysts are more frequently confounded with hydronephrosis (*vide infra*), and, in women, with ovarian tumors.

The prognosis is not wholly unfavorable. Permanent recovery has been repeatedly observed, especially after the rupture, or single or repeated evacuations, of the sac of the echinococcus; but, of course, echinococcus of the kidney may also be attended with numerous dangers, such as suppuration of the sac. The course of the disease is always very tedious.

A radical *treatment* is possible only by surgical means. Symptomatically, ice and local bloodletting are used when there are symptoms of local inflammation; and morphin, warm baths, and sometimes mechanical aids—e. g., the catheter—when there are symptoms of colic.

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[1] In regard to the general natural history of the echinococcus, compare p. 708.

2. **Distoma hematobium** (*Bilharzia hematobia—Blood-fluke*).—The distoma hematobium (see Fig. 99) is a parasite which belongs to the flukes or trematodes, and which occurs especially in Egypt, Abyssinia, and East Africa. Infection is said to take place partly by means of the drinking water and food, and partly, perhaps, by the parasite creeping into the urinary passages and the rectum during bathing. Natives are much oftener attacked than Europeans. The distoma has its chief abode in the branches of the portal vein, and particularly in the venous plexuses of the bladder and the rectum. Its eggs are often deposited in great numbers in the mucous membrane of the pelvis of the kidney, the ureter, and the bladder, and they excite in these places a severe inflammation with ulceration and consequent strictures, or the formation of concretions. Similar violent inflammation occurs, also, in the rectum and in the sexual organs. The course of the disease is very chronic. The main symptom is persistent hæmaturia. Many cases of so-called tropical hæmaturia are due to the distoma, and later we have the symptoms of local inflammation and of severe cystitis. The diagnosis may be established by the discovery of the eggs in the urine or the feces. Treatment is merely symptomatic.

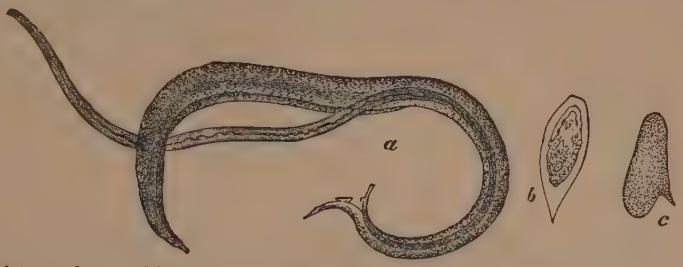


FIG. 99.—Distoma hematobium (from LEUCKART). *a*. Male and female, the latter in the gynecophorus canal of the former. Enlarged ten diameters. *b*. Egg with a terminal spine. *c*. Egg with a lateral spine. 150 diameters.

3. **Strongylus or Eustrongylus Gigas** (*Palisade Worm*).—This is a parasite occurring in the pelvis of the kidney in many animals—the dog, the wolf, the marten—and very rarely in man. In size and color it is not unlike an ordinary earthworm. It may produce symptoms of severe pyelitis, with hemorrhages and colicky pains. No well-established cases have been reported of late.

4. **Filaria Sanguinis. Chyluria.**—The blood filaria of man, belonging to the roundworms, has obtained a special clinical interest, since it is recognized, from the investigations of Wucherer in Bahia in 1868, and of Lewis in the East Indies in 1870, as the cause of the tropical chyluria and some allied diseases, such as lymph scrotum, elephantiasis Arabum, and chylous ascites.

The full-grown filaria, "*filaria Bancrofti*," a very thin worm, about 3 or 4 inches long, has been found only a few times in man. Its seat is in the larger lymphatics, where is gives rise to chronic stasis of the lymph with its consequences—chronic hyperplasia of the connective tissue, etc. In chyluria, the parasites are probably situated in the main branches of the thoracic duct—at any rate, in such a place that a stasis of the lymph ensues in the lymphatics of the bladder, or perhaps, in some cases, of the pelvis of the kidney and the other urinary passages. If the distended lymph sac ruptures, the lymph or chyle is poured out into the urinary passages and is evacuated with the urine. Since this process may be often repeated, the intermittent course

of chyluria is thus explained. The individual attacks of the disease may come on during years at intervals of weeks or months. They are often associated with pain and febrile symptoms.

The condition of the urine, which in many cases may look almost exactly like milk, is most characteristic. A creamy layer of fat forms upon the surface. If we shake the urine with ether, the greater part of the fat can be removed, and the urine rendered clear. The fat in the urine may amount to two or three per cent. The chyluria is often associated with a hematuria coming from the ruptured veins. The urine then looks bloody red, and shows under the microscope many red blood corpuscles besides the fat drops. Large clots often form in the urine.

Fig. 100.—(From Scheube.) Embryos of filaria.

The embryos of filaria, found in the urine in very many cases, although not in all, form the most important diagnostic feature in the urine. These (see Fig. 100) are objects $\frac{1}{12}$ to $\frac{1}{8}$ in. (0.2–0.3 millimeter) long, with a diameter about equal to that of a red blood corpuscle. They are usually imbedded in a very delicate sheath, which often projects at the end of the animal, and show a constant, vigorous vibrating motion. They have also been found in the blood of the patient, as well as in the urine, and, strange to say, especially during the night.

The course of the filaria disease may vary considerably. Many patients reach an advanced age; in others, severe general symptoms, like anæmia and emaciation, finally come on. The different forms in which the disease occurs —chyluria, elephantiasis, etc.—are combined in manifold ways.

The region of the geographical distribution of the disease lies almost wholly in hot countries. It has so far been most frequently observed in Brazil, the Antilles, the East Indies, China, Japan, Egypt, Cape Colony, and Australia. Nothing definite is yet known of the precise mode of invasion of the parasites. According to Manson's investigations, mosquitoes play an important part here. [This view is now well established.]

In regard to treatment, apart from any surgical interference, we may try picronitrate of potassium, 3 to 8 gr. (gm. 0.2 to 0.5), in pills or capsules, several times a day (Scheube).

CHAPTER X

MOVABLE KIDNEY (FLOATING KIDNEY, REN MOBILIS)

Ætiology.—With some practice the physician can frequently palpate the kidneys, particularly the right kidney, even under absolutely normal condi-

tions ("palpable kidney"). This is much oftener the case in women with lax and yielding abdominal walls than in men. With every inspiration the kidney is pushed somewhat downward. In many cases, therefore, the kidney, or at least its lower half, is felt only upon deep inspiration. If the kidney is not only palpable, but also capable in greater or less degree of being moved about by the hand, and if, consequently, we do not always find the kidney in the same place, then we describe it as an abnormally movable kidney; or, if the case is extreme, a floating kidney. What may be the causes of this extreme mobility of the kidney is not settled. Often it may be due to the congenital, anatomical relations of the peritoneum, and other tissues around the kidney. This is indicated particularly by the occasional occurrence of movable kidney in children. Certain external mechanical factors, however, undoubtedly play the most important rôle. First among these is the distention and relaxation of the abdominal walls as a result of pregnancy; and, secondly, the influence of clothing, waistbands, and corsets. This explains the fact that movable kidney is found mainly in women, and particularly in married women. At the same time, the abnormal mobility and downward displacement of the kidney are often merely a part of a general "enteroptosis" (see page 565), and are therefore associated with gastroptosis, coloptosis, and corset liver. That the movable kidney is far more often the right than the left is probably due to the fact that the right kidney is originally less firmly fastened, and is placed lower, and also that it is affected by the great mass of the liver which lies above it.

Numerous other causative influences have been mentioned, but none of them is as important as the preceding: they are, severe physical labor with frequent tension of the abdominal muscles, injuries involving the region of the kidneys, diseases of the neighboring organs, particularly displacement of the uterus, and, finally, emaciation, causing absorption of the fat which supports the kidneys and makes tense the abdominal walls. After death a movable kidney cannot be recognized unless it is found in an abnormal position—e. g., in front of the vertebral column, or close to the anterior abdominal walls. In such cases its external edge may be directed downward and its inner edge upward.

Symptoms.—The clinical importance of movable kidney is estimated very differently by different physicians. Many conceive that it is a very frequent cause of manifold abdominal disturbances; others are almost inclined to deny it any influence on the health. We ourselves believe that a movable kidney as such may, in fact, excite disagreeable symptoms, but that this is the exceptional occurrence. As we have already mentioned, palpability and slight movability of the right kidney are so frequent in women that, as soon as one's attention is directed to the matter, one finds this condition in a large proportion of all cases in which there is occasion to make a careful abdominal examination. The pains and abnormal sensations in the abdomen, of which women so frequently complain, are difficult of interpretation, and it is, of course, very seductive and convenient to refer them to a movable kidney; but if we consider how often we find an exactly similar movable kidney in women who are entirely free from abdominal symptoms, we cannot avoid suspicion as to the importance of the condition.

It is difficult to form a characteristic group of symptoms which may be

unhesitatingly referred to movable kidney. First, pain should be named. This is to a certain extent local, but it often radiates into the epigastrium and the sacral and lumbar regions, and sometimes is colicky. Not infrequently the pain is associated with nausea. The discomfort is aggravated by any decided motion of the patient, or by riding, driving, and the like; but during repose it becomes slight, or entirely disappears. In rare cases we have symptoms of strangulation. These occur periodically, and are said to be especially frequent at the time of the menses. They consist of the sudden onset of violent pain, chilliness, which seldom amounts to a rigor, great tenderness and tenseness of the abdomen, slight fever, vomiting, and collapse. During this time the urine is usually scanty, and the amount does not increase again till the attack is over, about three to five days. The precise cause of these symptoms is supposed to be a sudden damming up of the urine by a kink or twist in the ureter. This occasions acute hydronephrosis, and resultant phenomena, which last until the urinary channels are free again. In other cases an acute swelling of the kidney and distention of its capsule are said to arise from a kink in the kidney veins. We must openly confess that we have never observed a case of that character in which the symptoms could positively be interpreted as symptoms of strangulation of a movable kidney, and that the occurrence of the conditions in question has never seemed clearly proved to us.

The discovery of floating kidney by palpation is usually easy. The physician examines the patient lying on her back, applying his left hand on the right lumbar region and pressing forward, while his right hand presses from in front in the opposite direction. It is then often possible, especially upon deep inspiration (*vide supra*), to catch the kidney between both hands, and so form an opinion as to its position and mobility.

The next point is to determine whether, in any particular case, the symptoms which may be present are actually due to the movable kidney which has been discovered, or whether the latter is an unimportant matter. This question demands a careful and complete physical examination and a consideration of all the symptoms. In the first place, we should determine accurately the condition of the stomach (gastroptosis, etc.) and of the intestines (chronic catarrh of the colon, mucous colitis, etc.), and we should also think of the possibility of gallstones, which may produce a very similar group of symptoms. Gallstones are especially apt to occur in women with corset liver and enteroptosis, and this explains why a movable kidney is not infrequently found in cases of biliary colic, although it has nothing to do with the attack. With so-called "symptoms of strangulation," the possibility of renal calculi must be borne in mind.

In a great majority of cases of "floating kidney" we have to do with those familiar and frequent conditions of a "nervous" character which are termed hysteria or neurasthenia. Women of this kind suffer very frequently from all sorts of painful sensations in the abdomen, and from dyspeptic disturbances and the like, and of course we very often find in them a movable kidney. In such cases the abdominal symptoms are also of a nervous character, as can usually be easily determined from the general condition of the patient, from her psychical and cerebral symptoms, and the indications of a cardiac neurosis, and also from the results of suggestive treatment. Still, a certain number of cases may be very difficult of diagnosis. If we find a movable kidney in a

nervous, hysterical woman, it is not always advisable to apprise the patient of the fact, for with a person of this sort the mere idea of possessing a "floating kidney" is enough to stir up a host of subjective symptoms. On the other hand, it may be said that the floating kidney may serve as a very handy instrument for suggestive therapeutics. If we say to such a patient that her annoyance would immediately vanish after the application of a suitable bandage, we may sometimes attain great therapeutic success, although this is apt to be but temporary.

Treatment.—If we deem that the movable kidney requires treatment, the first thing to recommend is the wearing of a suitable support. The bandages, with special pads and the like, we regard as usually of no value, while a good elastic abdominal bandage, with thigh straps, or a well-fitted "abdominal corset," is sometimes decidedly efficient, particularly in all cases of general enteroptosis. We should also see that the patient is properly nourished. If she is emaciated, we should prescribe rest with the largest possible amount of food, so-called overfeeding. As more and more fat is deposited in the abdomen, the abdominal walls regain their tension and the kidneys receive better support. It is also advisable to sponge the abdomen with cold water or brandy, or to employ massage, the Scotch douche, or electricity.

If there are any symptoms of strangulation, we should, of course, put the patient to bed and order hot poultices and opium. We may also make a cautious attempt to replace the organ. If severe attacks of this sort recur, the possibility of surgical treatment should be considered (nephrorrhaphy or nephropexy). In a fairly large number of cases the patients are said to have been entirely freed of their discomfort by this method. We doubt if all these cases would stand the test of a rigid criticism. It is certain that the operative result is often of a purely suggestive nature.

APPENDIX

THE DISEASES OF THE SUPRARENAL CAPSULES AND ADDISON'S DISEASE (BRONZED SKIN)

Ætiology and Pathological Anatomy.—In the year 1855 the English physician Addison published for the first time a list of cases in which, besides the symptoms of a general bodily weakness and anæmia, a peculiar dark pigmentation of the skin had gradually developed. Since disease of the suprarenal capsule was found at the autopsy in all cases, Addison concluded that this was the immediate cause of the bronze coloring of the skin. Observations similar to Addison's were soon made in greater numbers, so that the fact itself cannot be doubted.

The actual nature of Addison's disease is, to be sure, by no means entirely cleared up. Still, through the newer discoveries regarding the physiological significance of the suprarenals, our conceptions of the disease have become decidedly more intelligible. All the more so as the disease is obviously correlated to quite a number of morbid processes, which likewise depend upon a disturbance in the so-called internal secretions (the suprarenal bodies, thyroid gland, thymus and pituitary glands).

The suprarenal capsules are composed of two entirely different tissues, the medullary and cortical substances. Their anatomical intermingling, however, speaks, in all probability, for a close mutual physiological relationship. The cortex is composed of epithelial cells with a profuse network of thin-walled capillaries. The medulla, on the other hand, is certainly a part of the sympathetic system. Accordingly, it consists for the smaller part in typical sympathetic ganglion cells and numerous nonmedullated nerve fibers, and for the greater part in so-called chromophile cells. These are peculiar, large branching cells, which take on a characteristic brown stain with chromium salts. They accompany the sympathetic nervous tissue all over the body, and are also found in other accessory organs of the sympathetic system (carotid glands, coccygeal gland, etc.). Adrenalin (suprarenin) is found only in the extract of the medullary portion, and is derived from the chromophile cells. The remarkable action of this substance in raising the blood pressure was first accurately studied by Oliver and Schafer in 1884. The so-called accessory adrenals are, as a rule, composed of only cortical substance.

Brown-Séquard, in 1856, was the first to demonstrate that the suprarenal capsules were absolutely essential to life. He found that the extirpation of both suprarenals led in a short time to the death of the animal experimented upon. Through numerous observations in recent years the following phenomena have been established as the main results of suprarenal extirpation: General muscular weakness, nervous disturbances (somnolence, coma, epileptiform seizures, dilatation of the pupil, etc.), fall of blood pressure, abnormal pigmentation, lowering of the body temperature, general disturbances of nutrition, etc. These symptoms are explained partly by the loss of the neutralizing function of the suprarenals for certain poisons in the body, and partly by the loss of the secretory function of the organ. The former function is supposed to reside mainly in the cortex, while the latter, as already mentioned, is exercised chiefly by the medulla. The main results of the physiological action of suprarenal extract (adrenalin) thus far established are as follows:

1. Marked increase in the blood pressure due to contraction of the smaller vessels, as a result of direct peripheral irritation.
2. Strengthening of the cardiac contraction and slowing of the pulse.
3. Slowing of the respiration, which, at the same time, becomes more superficial.
4. Dilatation of the pupils. This can also be demonstrated in the enucleated eyes of frogs.
5. Inhibition of intestinal peristalsis, etc.

We are still far removed from a complete explanation of the clinical phenomena of Addison's disease on the basis of these physiological facts. Still, quite a number of connecting links between the results of experimental and clinical observation must be recognized. For the time being we must, however, study from a clinical standpoint the symptoms which point toward a disturbance of the function of the suprarenal bodies in a diseased individual. Even Addison himself pointed out the fact that the actual nature of the lesion in the suprarenals was by no means always the same. Hence the disease named after him is, first of all, to be regarded not as a definite pathological condition, but far more as a peculiar symptom-complex due to a loss of supra-

renal function. With comparative frequency tuberculosis of the suprarenal capsules is found to be the basis of the Addison symptom-complex. The suprarenals are then either enlarged and infiltrated with caseated tuberculous tissue, or they are in part already cicatrized and contracted. Almost always other tubercular disease is found coexistent at autopsy, especially caseation of the mesenteric lymph-glands, pulmonary tuberculosis, etc. Besides tuberculosis, other morbid processes, such as neoplasms, etc., may sometimes be found in the suprarenals. Recent personal clinical and pathological observations have convinced us that there exists a special peculiar form of Addison's disease in which at autopsy only an extreme atrophy of the suprarenal bodies is found. In these cases both suprarenals are converted into thin parchment-like structures, in which the actual parenchyma has almost entirely degenerated. In somewhat the same manner in which we look upon symptomatic glycosuria as compared to true diabetes, so, in our opinion, must we regard the symptoms of Addison's disease developing in other diseases of the suprarenal bodies as a secondary symptom-complex, in comparison with primary suprarenal atrophy with the fully developed clinical picture of Addison's disease. Only these latter cases do we regard as true primary Addison's disease. To be sure, we know nothing regarding the causes of this apparently primary suprarenal disease. Impaired nutrition, psychic influences, traumatism, possibly sometimes play a certain rôle. In the main, however, the disease is probably due to direct exogenous or endogenous harmful influences acting upon the specific tissue of the organ. The disease itself is rare, and occurs somewhat more frequently in males than in females. Although usually a disease of middle life, we ourselves have even seen a case in a ten-year-old child.

Of the lesions in other organs we must also mention that Peyer's patches and the solitary follicles of the intestine are, as a rule, swollen. The spleen is somewhat enlarged in some cases, but not in others. There is no striking pigmentation of the internal organs. Still, in one very characteristic fatal case we saw marked pigmentation in the internal lymph-glands and in the liver, and once, besides, in the retina, during the lifetime of the ten-year-old male patient. The changes in the skin and in certain mucous membranes will be mentioned below.

Symptomatology.—The purest type of Addison's disease appears in those cases mentioned above where the symptoms are apparently primary in their development, and do not come on in the course of some other disease, such as phthisis or cancer.

The first symptoms of the disease are usually of a general nature, and are referable to a gradually increasing anæmia and to general weakness and physical lassitude. The anæmia shows itself objectively through the pallor of the skin and the diminution in the number of red blood corpuscles, but without other definite anomalies of the blood that can be made out. A relative lymphocytoses is often present, and perhaps deserves mention. There is no increase of the leucocytes. The general muscular weakness (asthenia) is one of the most regular symptoms of the disease. This is shown less in inability to make a few vigorous contractions than in extremely rapid exhaustion of the separate muscles. As a consequence, any long-continued muscular exertion, such as walking for any length of time, standing, continuous labor, becomes very difficult. Not infrequently the patients complain of rheumatoid joint and muscle

pains. To these are added numerous other so-called nervous symptoms. Among these are the mental torpor and lack of energy, the headache and tendency to syncope, the tinnitus aurium, the insomnia or constant sleepiness, etc. Sometimes there are marked psychical disturbances, such as impairment of memory, feebleness of mind, or exaltation. The patient's general nutrition often suffers very considerably; but it must be added that in Addison's disease, as in other anæmias, the fatty layer, especially over the abdomen, often remains remarkably well developed.

In women persistent amenorrhea often develops. Besides the symptoms which have been enumerated, there is often gastric disturbance. The secretion of the gastric juice is interfered with, and frequently free hydrochloric acid and pepsin are absent. The appetite is poor, and there is very often vomiting. The latter may sometimes be almost uncontrollable, and then it is one of the most distressing symptoms of the disease. It is usually due not to an anatomical change in the stomach, but probably to nervous or toxic influences. In one severe case we saw a constant and distressing hiccough. Cardialgic symptoms are also frequent. The bowels are sluggish, as a rule, but there is sometimes diarrhea. We sometimes hear functional murmurs in the heart, but, as a rule, its sounds are pure, though feeble. The pulse is usually moderately accelerated.

Recently, in view of the physiological action of adrenalin, special attention has been devoted to the state of the blood pressure. In general, it may be said that the blood pressure is abnormally low, and that the pulse is therefore small, soft, and easily compressible. The liver appears to remain normal. The spleen is often somewhat enlarged. We have already referred to the frequent swelling of the abdominal lymph-glands, which, however, is not clinically prominent. The liver and spleen do not show any special changes. Albuminuria is exceptional, and depends upon complications, such as amyloid kidney. Occasionally, large amounts of indican, urobilin, etc., appear in the urine without their being any special significance to these findings. The bodily temperature is not elevated, and not infrequently, indeed, it is decidedly subnormal.

The special characteristic symptom, which alone renders the diagnosis possible, is the gradual onset of a peculiar pigmentation of the skin. This usually shows itself first in the face and on the backs of the hands, and also in those parts which normally present a greater pigmentation (the areola of the nipples, the axillæ, and the genitals), or which are exposed to greater pressure by the clothing, as the hips and shoulders. The white palms of the hands not infrequently form a marked contrast with their darkly pigmented dorsal surfaces. There is, however, also a marked pigment deposit in the flexion folds of the fingers. After numerous personal observations, we have come to regard this early peculiar dark pigmentation of the folds of skin on the flexor side of the fingers and palms as having a certain diagnostic significance. It is especially noteworthy that dark pigmented spots and stripes usually develop on the mucous membrane of the lips, particularly at the corners of the mouth, on the gums, the palate, and inside the cheeks. We also consider the brown pigmentation of the edges of the conjunctivæ of the lower lids quite characteristic. The intensity of the coloring differs in different cases. It usually increases as the general condition grows worse. In the most intense cases

almost the whole skin may become dark brown or black, like that of a mulatto or negro. Sometimes, however, the pigmentation remains limited to separate large or small spots, and in other parts of the skin there may then be even a marked loss of pigment. The hair is usually not changed. The pigmentation of the skin usually increases during the whole disease; only exceptionally does the skin become light again in the later stages. Other changes in the skin are usually absent.

The real cause of the accumulation of pigment in the skin is wholly unknown. We find on microscopic examination of the skin that the pigment lies not only in the cells of the rete Malpighii, but also in the corium, especially along its blood vessels. It is probably formed from the blood pigment, and is carried by wandering cells from the cutis into the epithelial layers of the skin (Demieville, Nothnagel). The pigment in Addison's disease does not contain iron. It must furthermore be mentioned that the skin pigmentation in Addison's disease, although of great diagnostic significance, must not be regarded *per se* as an absolutely essential symptom. The variation of the relationship between the degree of the skin pigmentation and the severity of the other symptoms is in itself worthy of note. It is quite possible also that cases of Addison's disease (*i. e.*, suprarenal disease) may exist without skin changes. These cases as yet are not capable of diagnosis.

The course of Addison's disease is almost always chronic, and may last for years, but cases have been described with a rather acute course. The disease sometimes begins with violent initial febrile symptoms, vomiting, and diarrhea. The disease then has a comparatively rapid termination after a few months, or a second chronic stage may follow the first acute one.

The final termination of Addison's disease is always unfavorable. Temporary remissions are often observed, but the disease always becomes worse again after them. Death usually ensues gradually amidst the signs of increasing general uræmia and weakness. In some cases severe nervous symptoms also come on toward the end of the disease—coma, delirium, or epileptiform attacks. Conditions of this sort may develop comparatively suddenly and unexpectedly; indeed, they can be explained only on the hypothesis of toxic influences, and remind us of diabetic or uræmic coma.

The clinical picture of secondary Addison's disease is composed of the same symptoms, only they are added to those of an already existing affection. This complication is most frequently observed in tuberculosis, where, as has been mentioned, it is usually traceable to a tuberculous disease of the suprarenals. It may also occur in carcinoma. The development of symptoms of Addison's disease in hypernephroma has already been mentioned (page 795). Finally, it is very interesting to note that symptoms of Addison's disease occasionally occur in association with exophthalmic goiter, as well as with scleroderma, etc. In all these cases, it is probably always the abnormal pigmentation of the skin that first calls attention to the remarkable complication.

Treatment.—Organotherapy has been repeatedly tried in Addison's disease, on the same lines as the treatment of diseases of the thyroid gland. The adrenals of the sheep have been administered, either as fresh glands, finely minced and eaten with bread, or as an extract, or in the form of tablets of the dried glands. In some cases good results seem to have been obtained in this manner, even to diminution in pigmentation, but in many other cases this

prescription has no beneficial effect. We ourselves administered adrenal tablets in one case for a long time without any advantage. Still, it would be proper to continue these trials. Whether treatment with Koch's tuberculin might cause permanent benefit in those cases of symptomatic Addison's disease which are due to tuberculosis is as yet doubtful. A cautious trial might be made under proper circumstances. As to other remedies, tonics are generally employed—nourishing food, iron, quinin, and arsenic; iodid of potassium, bromid of potassium, and electricity have also been tried, but without success. Symptomatic treatment is needed for the vomiting, diarrhea, and nervous attacks. Experience has shown that great caution should be exercised in prescribing laxatives, because in repeated instances such remedies have had decidedly unfavorable results.

SECTION II

Diseases of the Pelvis, of the Kidney, and of the Bladder

CHAPTER I

INFLAMMATION OF THE PELVIS OF THE KIDNEY—PYELITIS

Ætiology.—Isolated primary pyelitis does not often occur as an independent disease. It is, however, more common than was formerly believed, and acute primary pyelitis, as well as acute pyelocystitis, has been repeatedly observed, not only in adults, but also, and especially, in children. In the majority of cases the exciting cause is an infection of the urinary passages with the colon bacillus, and it is not improbable that the infection originates from the intestine. It has not, however, been definitely decided whether the bacilli pass directly from the intestine into the urinary passages, or whether they first enter the blood stream, and thence gain entrance to the urinary organs. Secondary pyelitis is more common than the primary form. It is either a complication or a result of other diseases, and in such cases often attracts but little clinical attention.

We sometimes find a rather moderate pyelitis in the bodies of persons who have died of severe general infectious diseases—typhoid fever, smallpox, diphtheria, or pyæmia. It has not been positively determined in these cases how far chemico-toxic influences play a rôle, and how far the organized infectious agent itself must be taken into consideration. In typhoid pyelitis, the typhoid bacilli, which are often excreted in great numbers with the urine, probably have a directly harmful action upon the mucous membrane of the urinary passages. It is worthy of mention, too, that in secondary pyelitis colon bacilli have often been found in the urine. Of the special poisons which may produce a pyelitis, cantharides and certain balsamic substances, such as copaiba balsam, are, above all, to be mentioned.

Pyelitis very often rises from a direct extension of inflammation from the neighboring organs. In many cases of acute and chronic nephritis the pelvis of the kidney takes part in the inflammation to a greater or less degree; but

an ascending extension of the inflammation from primary diseases of the urethra or bladder is still more common. Any case of urethritis or cystitis may, if it lasts long, advance upward to the ureters and the pelvis of the kidney, so that in severe cases we often find an inflammation of the whole urinary tract, a pyelocystitis, and even a "ureteritis." It has been already mentioned (page 789) that the inflammation may extend still farther to the kidneys themselves (pyelonephritis), and we shall refer to this repeatedly. Of all these varieties of ascending inflammation in the urinary passages, none is so frequent or of so great practical importance as that which results from persistent narrowing of the urethra (stricture, hypertrophy of the prostate), and the consequent obstruction to the flow of urine. We shall revert to this important variety when discussing hydronephrosis. Very frequently the ascending pyelitis is a sequel to cystitis in disease of the spinal cord, with paralysis of the bladder.

Another form of pyelitis is that caused by the presence of foreign bodies in the pelvis of the kidneys—e. g., calculous pyelitis due to the mechanical irritation of renal calculi. This also will have a special consideration below. Much less frequent causes are retained blood clots, parasites (*vide supra*), and genuine foreign bodies.

Pathological Anatomy.—In simple catarrhal inflammation the mucous membrane of the pelvis of the kidney is reddened, swollen, and covered with an abundant secretion, which contains varying amounts of pus corpuscles and epithelium. In severer inflammations we often find quite numerous little hemorrhages in the mucous membrane, and sometimes little gray nodules, corresponding to swollen lymph-follicles.

In severe cases, which are seen almost solely as a complication of a more extensive affection of the urinary passages, such as pyelocystitis, we have a purulent, ulcerative inflammation, which may even assume a diphtheritic character. In these cases the kidneys are almost always coincidently involved —pyelonephritis. If the nephritic abscesses break into the pelvis of the kidney, there arises an ulcerative destruction of the renal tissue, so that the pelvis of the kidney is filled with pus and bounded by extensive ulcers, which often penetrate deeply into the substance of the kidney—pyonephrosis. The pyelonephritic abscesses reaching to the outer surface of the kidney, and usually giving a striated appearance to a section of the organ, have already been described (see last section) and their bacterial origin mentioned.

The condition differs when the kidney is involved, as in many cases of chronic pyelitis. This appears most frequently as a result of retention of urine, and hence it is usually associated with a dilatation of the pelvis of the kidney. In these cases we sometimes find pronounced processes of contraction in the kidneys—that is, a partial atrophy of the renal tissue, increase of the interstitial connective tissue, and evident cicatricial depressions on the surface—in a word, a secondary contracted kidney, arising as a result of pyelitis, which differs from genuine contraction of the kidney only in its ætiology.

Clinical Symptoms.—Since in most cases pyelitis develops only as one symptom of a more extensive morbid process, its clinical symptoms are usually but slightly prominent in the general course of the illness. In what follows, therefore, we cannot give any complete description of the clinical course of pyelitis, but we must mention only those symptoms from which,

when there is an affection of the urinary passages, we may conclude that the pelvis of the kidney takes part in the morbid process.

The most essential sign which the urine presents in all inflammatory affections of the urinary passages, the presence of mucus and pus, will be described more fully in the chapter on cystitis (*vide infra*). In pyelitis, also, the mucopurulent secretion of the pelvic mucous membrane must mix with the urine, and in every severe purulent inflammation the amount of pus in the urine must be considerable. We can never decide with certainty, from the mere presence of pus in the urine, as to the place where the pus mixes with the urine, whether in the pelvis of the kidney or in the bladder, or even in the urethra. If only we were able to demonstrate other morphological elements besides the pus corpuscles, so characteristic that their origin might be affirmed with certainty to be the pelvis of the kidney, we might, by means of them, make our diagnosis of pyelitis absolute. Unfortunately, however, the microscopic sediment of the urine leaves much in this regard to be desired. The greatest weight was formerly ascribed to the discovery of pelvic epithelium; especially the triangular, long-tailed epithelial cells, sometimes arranged one upon the other, like the tiles of a roof (see Fig. 101), were regarded as an indication that the pelvis of the kidney was involved in the inflammation; but the diagnostic significance of these cells is very slight, for, on the one hand, they may be absent in a severe case of pyelitis, and, on the other hand, precisely similar epithelium comes from the mucous membrane of the bladder. Greater value is given of late to the discovery of certain casts from the mouths of the urinary canaliculi (*ductus papillares*) —structures which are almost invariably involved in all but the milder cases of pyelitis. Tubuliform epithelial casts, cylindrical formations composed of pus corpuscles, and, above all, casts composed of micrococci, have been repeatedly found in the urinary sediment in cases of pyelitis, and possess some diagnostic importance (Fürbringer and others).

Fig. 101.—Epithelium from the pelvis of the kidney.

With regard to the other characteristics of the urine, it should be mentioned that it is often remarkably abundant in pyelitis, and that it then of course is pale and has a comparatively low specific gravity. The reaction of the urine is usually acid, despite the admixture of pus. That this acid reaction is an effectual distinction from the urine of cystitis cannot be maintained (*vide infra*). We may, however, say that the tendency of the urine to ammoniacal fermentation is decidedly greater in cases of cystitis than in cases of pyelitis. The amount of albumen in the urine corresponds to the amount of pus. If there is a very large amount of albumen, it arouses suspicion of a coincident nephritis. The only decisive proof of such a complication lies in the finding of genuine urinary casts. In cases of simple pyelitis it is exceptional for the urine to be bloody, while in calculous pyelitis (*q. v.*) blood is often present.

Besides the quality of the urine, another symptom is pain in the region of the kidneys, occurring either spontaneously or through pressure. This symptom is present in many cases of pyelitis, and is therefore of diagnostic

importance. The pain is often very severe, and radiates along the ureters to the bladder. In some cases, however, there may be no pain at all, so that while its presence is one symptom of pyelitis, its absence does not weigh against the disease.

All the other symptoms may be directly dependent upon the pyelitis, but they may usually be referred in great degree to the other coexisting affections. First among these is fever, that either shows an irregularly remitting course, or appears in single high elevations of temperature, usually associated with rigors. The fever, however, seldom shows this latter pyæmic character except in the severe purulent forms, where we usually also have the formation of renal abscesses—that is, a pyelonephritis. Besides the fever there are often, in severe cases, general nervous symptoms, such as headache, delirium, and sopor. These symptoms are to be referred partly to the general pyæmic infection of the body and partly, as well, to the absorption of ammonia from the decomposing urine into the blood—the "ammoniæmia" of Treitz and Jaksch.

The whole course of pyelitis differs so much according to the primary disease present that nothing of general application can be said about it. The milder forms, which often pass off rapidly, are found most commonly in childbed, and sometimes in acute infectious diseases, poisonings, and as a result of mild cystitis. The severe forms are chiefly, as we have said, pyelocystitis and pyelonephritis, as a result of strictures of the urinary tract (*vide infra*), of severe cystitis in diseases of the spinal cord, and in other severe diseases of the kidney and of the pelvis of the kidney, such as new growths and parasites. They usually constitute a very tedious and incurable affection, which lasts until the patient's death.

What appears to be a primary pyelitis occurs chiefly in children and young persons, and often begins with the general manifestations of an acute febrile infection. At first the diagnosis of the true condition is difficult, but is made possible by pains in the region of the kidneys, and, above all, by the presence of pus in the urine. The absence of bladder tenesmus and of frequent micturition is to be noted in contradistinction to the prominence of these symptoms in every case of severe cystitis. Coincident gastrointestinal symptoms are often observed. With proper care and treatment, a cure generally results in from eight to fourteen days.

The implication of the kidneys is shown by the presence of casts in the urine in addition to the pus corpuscles. In the cases above mentioned, in which a chronic pyelonephritis is complicated with the contracted kidney, the condition of the urine is the same in many respects as in genuine contracted kidney. It is abundant, usually has a low specific gravity, and contains, besides the pus corpuscles, a few short hyaline casts. In such cases there may be developed a secondary hypertrophy of the left ventricle, unless the general nutrition of the patient is too much impaired.

Great progress in the more exact diagnosis of diseases in connection with which pyuria occurs, is due to cystoscopy and to separate examination of the urine from each kidney, made possible by the catheterization of the ureters, and by other methods of segregation. Detailed information in regard to these important methods of examination will be found in special works upon the subject.

Treatment.—The treatment of pyelitis coincides mainly with the treatment of the primary disease—for example, renal calculi—and to that extent it needs no detailed description here. In regard to the general dietary, the copious drinking of fluids (mineral waters of Wildungen, Fachingen, Obersalzbrunnen, etc.) will usually be found useful. A diet in which milk predominates is, therefore, frequently prescribed with success. Spices and stimulating foods are to be avoided. Among the internal remedies to which a favorable influence is ascribed in connection with affections of the urinary tract, we have, first of all, the astringents (tannin, tannigen, etc.). Their therapeutic effects, however, are usually very slight, and often they are ill borne. Certain antiseptic remedies are preferable, particularly salol in capsules of 8 gr. (gm. 0.5) three to five times a day; also boric acid, 5 to 8 gr. (gm. 0.3 to 0.5) three times daily, camphoric acid and chlorate of potassium. Urotropin, 8 to 15 gr. (gm. 0.5 to 1) several times daily, is the remedy at present most frequently employed, particularly in cystopyelitis combined with bacteriuria or with fermentation of the urine. Judging from personal experience, the results are most praiseworthy. Helmitol is another drug with a similar action.

Local applications to the region of the kidneys, warm poultices, or exceptionally local bloodletting, are indicated only when there is severe pain, and then, of course, narcotics must also be used under some circumstances. In this respect warm baths also do good service at times.

CHAPTER II

NEPHROLITHIASIS

(Renal Calculus. Renal Gravel. Pyelitis Calculosa)

Occurrence, Chemical Composition, and Ætiology of Renal Concretions.—The precipitated concretions of the urinary constituents which form in the pelvis of the kidney, and which, under some circumstances, may be passed from it with the urine, are designated, according to their size and nature, as renal sand, a fine, pulverized precipitate; renal gravel, gravel-like, granular concretions about the size of the ordinary coarse grains of sand, which can usually pass through the ureters without special difficulty; or renal calculi, the larger concretions. The last are about the size of a millet seed or a pea, but larger stones are occasionally seen which may even resemble actual casts of the pelvis of the kidney. We usually find a calculus in only one kidney, although both kidneys may be affected.

In regard to the chemical nature of renal concretions, they consist most frequently of uric acid and urates. The uric-acid stones are hard, have a brown-red or blackish color, and a crystalline fracture, which in large stones is usually plainly laminated; and, on the whole, a smooth although irregularly shaped surface. If a small portion of the stone is pulverized and the powder evaporated to dryness with a little concentrated nitric acid, a red spot arises, the color of which changes to violet on the addition of caustic soda (the so-called murexid test for uric acid.) More rarely the renal concretions con-

sist of calcic oxalate. The oxalate calculi are extremely hard, have a dark-brown, almost blackish, color and a rough surface, often furnished with many prickles, from which reason they are often called "mulberry calculi." Their fracture sometimes shows a radiated but never a laminated arrangement. Stones are also frequently seen, which consist of alternating layers of uric acid and calcic oxalate, or which have a nucleus of uric acid and a coating of calcic oxalate. The phosphatic calculi are another variety of renal concretions. We only rarely have to do, however, with stones which consist exclusively of basic calcic phosphate or ammonio-magnesic phosphate, but we oftener have secondary deposits of layers of phosphate which are precipitated on uric acid or mulberry calculi in urine which has become alkaline. The pure phosphatic calculi, of course, generally with a slight admixture of calcic carbonate, are grayish-white and rather soft, so that they can be crushed with the finger. The largest specimens of this kind are not found usually in the pelvis of the kidney, but in the bladder; still, as we have ourselves observed, large and pure phosphatic calculi do occur in the pelvis of the kidney, without any uric-acid nucleus. All the other calculous formations are so rare that they scarcely possess clinical interest. We may mention the light-yellow cystin [1] calculi with a surface of waxy luster, the xanthin calculi, and the indigo calculi.

As to the precise causes of all these concretions we have as yet no certain knowledge. The uric acid originates from the albuminous substances of the nuclei of the cells, the so-called nucleins. An increase in the destruction of leucocytes seems to be associated with an increased excretion of uric acid. The formation of a solid deposit of uric acid is usually associated with marked acidity of the urine. Yet, we do not know at all in what way these circumstances bring about the development of renal calculi. There is much probability in the assumption that some solid substance or other usually forms the nucleus, and gives the impulse to the formation of at least the larger renal calculi. Such nuclei may be coagulated mucus, bits of epithelium, and perhaps bacteria. It is an interesting fact, although we cannot completely explain it that, as Meckel, Ebstein, and others have shown, the microscopic examination of minute scales of calculi disclose the crystalline uric acid deposited in a supporting structure of an albuminous character. With regard to the formation of oxalate calculi, likewise, we possess no exact knowledge. Oxalic acid in the urine probably is due in part to the oxalic acid which is introduced into the system in vegetables, and in part is perhaps formed from the decomposition of albuminous substances. The deposits of crystals of calcic oxalate, as well as uric-acid crystals, from acid urine is well known to be a very frequent occurrence, while the actual formation of calculi is, as has been said, comparatively rare. The cause of the deposit of phosphatic concretions must lie in the urine becoming alkaline. In such cases, therefore, the development of concretions is probably preceded by disease of the pelvis of the kidney and the ingress of bacteria capable of exciting an alkaline fermentation of the urine.

In regard to the predisposing causes of calculous formation we must mention, first of all, that stones are often found in children, and next in frequency in advanced life. Men show a greater disposition to renal calculi than women.

[1] Cystin is a product of the splitting up of albuminous substances, and is usually further decomposed in the body. In some families, cystinuria occurs as a peculiar anomaly of tissue metabolism.

Heredity also seems to play a certain part, since the disease has been repeatedly observed in different members of the same family. The many relations which have been imagined between the formation of calculi and certain conditions in the manner of life and in the food taken, all lack definite proof. As to mode of life, the chief blame is laid upon an excessive meat diet, drinking copiously of new sour wines, and drinking water containing lime. It is remarkable that calculi are much more frequent in some countries (England, interior Russia) and regions than in others. In regard to the interesting but still entirely incomprehensible relations between the formation of renal calculi and other anomalies of tissue metabolism, especially gout, compare the chapter on Gout.

The Anatomical Changes Caused by Renal Calculi.—The presence of concretions in the pelvis of the kidney very frequently, but not always, excites pyelitis. This may exhibit all degrees, from a simple catarrhal inflammation to a diphtheritic or severe purulent inflammation of the pelvic mucous membrane. As a result of the mechanical irritation, there are quite frequently large or small hemorrhages.

If a severe purulent pyelitis has developed, this may bring with it all the sequelæ with which we have previously become acquainted. In severe cases the process may involve the kidneys, when there arises a pyelonephritis, with a purulent breaking down of the renal tissue, and, under some circumstances, even a perinephritis, with extensive suppuration in the vicinity of the kidney, and with occasional perforation into the neighboring organs. If the renal calculi have previously passed outward, they are not found at the autopsy, although they form the special starting point of the disease. Sometimes, however, the pus cavity is entirely filled with calculi.

A second important sequel of a renal calculus, which sometimes develops, is hydronephrosis (*vide infra*). It arises when a large stone blocks the passage from the pelvis of the kidney into the ureter, or when a smaller stone remains fast in the ureter and completely shuts off the passage of the urine. In the latter case there may also arise a pressure necrosis and perforation of the ureter. It goes without saying that inflammation and hydronephrosis or pyonephrosis may be combined.

Clinical Symptoms.—If there is merely the formation of renal sand or renal gravel in the urinary tract, this condition is sometimes associated with no symptoms at all. The little granules are washed away by the urine and evacuated, and at most they may give rise to slight pain in the region of the kidney and in the abdomen. Larger stones, however, may sometimes be wholly, or almost wholly, without symptoms, if their position and their smooth surface are such that they are comparatively harmless.

The characteristic clinical symptoms of nephrolithiasis do not appear until the results of mechanical irritation of the pelvis of the kidney arise, or until there is an incarceration of a calculus in the ureter. It is the latter circumstance which, after the analogy of gallstones, causes the most important symptom in the diagnosis of renal calculi—the pain, the so-called renal colic. Such an attack of colic sometimes comes on quite suddenly and unexpectedly; in other cases it is produced by some exciting cause—jumping, running, walking, or riding. The pain often becomes frightful; it radiates from the back and the lateral portions of the abdomen along the course of the ureters downward, and often upward, spreading particularly toward the bladder, testicles, and

thighs, and also up the back. A place in the abdomen which corresponds with the bend of the ureter, and on the right side almost with McBurney's point (page 594), is particularly often sensitive to pressure. Noteworthy also is the frequent sensibility to pressure of the testicle on the corresponding side. Nausea and repeated vomiting often occur. The bowels are constipated, the intestinal gases cannot be passed, and consequently the intestines become slightly distended and sensitive. The pulse is accelerated and often abnormally tense, and the temperature usually slightly elevated. Chills are rarely observed. In severe attacks there may be a general state of collapse with a small, rapid pulse, cold sweat, and attacks of fainting. The urine, as a rule, is scanty, but frequently evacuated with painful tenesmus. Sometimes it is almost entirely normal in quantity and composition, if it comes exclusively from the other free kidney. Oliguria, or even complete anuria, with its fatal consequences, invariably sets in if both ureters be occluded. Still, even with a unilateral calculous obstruction, the passage of urine may be scant or cease almost entirely as a result of a reflex inhibitory influence in some unknown way affecting the secretion of urine in the other kidney. Almost complete anuria has frequently been observed as long as eight days in cases of unilateral obstruction due to renal calculus. In such cases there is danger of uræmia, though it is astonishing how rarely this sets in in obstruction by stone. If, at the same time, there is a mechanical irritation or an inflammatory process, the urine often contains blood and pus. Even if the urine appears macroscopically to be normal, microscopic examination of the centrifugalized specimen often shows some leucocytes, red blood corpuscles, isolated hyaline casts, and particularly often, crystals of uric acid. The duration of the renal colic depends on the duration of the incarceration; it may last for a few hours or for several days. The attack often ends with the expulsion of the stone.

In the intervals between the several attacks some patients feel quite well, while others have slight pains in the loins and back, at times also distinctly localized in the region of the one kidney, as well as gastrointestinal disturbances; frequently urinary tenesmus exists. More serious symptoms set in as a result of severe mechanical irritation of the pelvis of the kidneys and of inflammatory complications. The pains in the regions of the kidneys then increase. The urine shows an admixture of pus, and contains pelvic epithelium and often blood. The frequent appearance of blood in the urine, in consequence of mechanical lesions of the mucous membrane of the pelvis of the kidneys, is a characteristic symptom of pyelitis calculosa. If we find, as sometimes happens, the urine at many times perfectly clear and normal, but at other times purulent, we may suspect an occasional blocking of the ureter coming from the diseased kidney by a renal calculus.

The symptoms are much more severe if the trouble goes on to a severe purulent pyelitis and pyelonephritis. We need not describe the details again here—the pain, fever, swelling, and perforation internally or externally—since they agree completely with what has been said before (see the previous chapter and Chapter VI in the previous section). A special chapter will be devoted to the symptomatology of hydronephrosis.

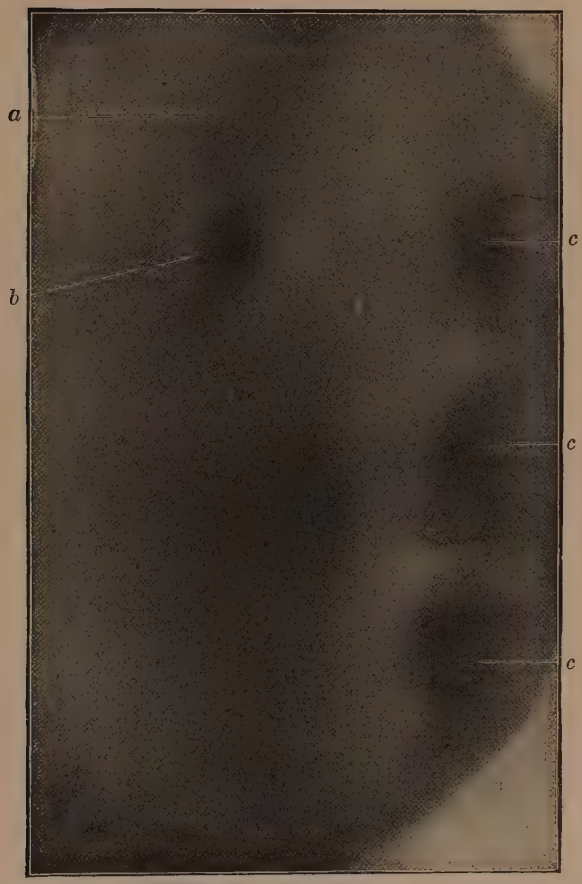
The course of nephrolithiasis is, as a rule, very tedious. Since the disposition to the formation of calculi usually persists, and since also the sequelæ which have once developed may last for a long time, a very chronic state often

develops, which, in varying ways and with manifold exacerbations and remissions, is composed of attacks of colic, hemorrhages, and symptoms of pyelocystitis.

In many cases, of course, complete recovery may finally ensue. The calculi present are passed, new ones are not formed, the pyelitis that has arisen disappears, and all the morbid symptoms cease; but, on the other hand, nephrolithiasis entails a number of dangers which seriously threaten life. These are, besides the rare occurrence of uræmia, first of all the development of pyelonephritis and of still more extensive suppurations, and a general decline in strength, pyæmic states, etc. There is also a possible danger in such chronic suppurations that a general amyloid degeneration of the internal organs may ensue. With regard to complications on the part of other organs, there is one point of special interest—viz., that sometimes gallstones and renal calculi are found in one and the same patient. Attention is again to be called to the combination of renal calculi with other gouty affections (*vide* chapter on Gout) as well as other constitutional diseases (diabetes, arteriosclerosis).

Diagnosis.—If typical attacks of renal colic with the passage of stones have repeatedly appeared, or if the attacks of colic are combined with the pronounced appearance of blood and pus in the urine, the diagnosis of nephrolithiasis presents no difficulties. It is different in the cases in which stones have never passed, and the patient complains only of pains, gastrointestinal disturbances, etc. In such cases, it is important that in addition to the other affections to be taken into consideration, the possibility of renal calculi be kept in view from the first, and the history taking and physical examination be directed accordingly. It is particularly difficult to distinguish renal colic from gallstone colic, recurring appendicitis, intestinal colic, purely nervous disturbances, floating kidney, lumbago, etc. The assumption of nephrolithiasis is based above all on a careful consideration of the character of the pains (radiation to the bladder and genital organs), sensitiveness to pressure in the region of the kidney and along the ureters, the frequent desire to urinate, with oliguria, increased tension of the pulse, and, above all, on the careful microscopic analysis of the urine (blood and pus cells, a few hyaline casts, uric-acid crystals). Very often, of course, a positive diagnosis demands continued observation of the case.

It is of great importance, particularly with regard to any operative treatment, to determine which kidney is the seat of the trouble, and whether one kidney, at any rate, is perfectly healthy or not. In considering these questions we must be guided by the chief seat of the pain; the appearance of the urine—for instance, observing whether there is an increase in the amount of pus if pressure is made upon the diseased kidney, and whether from time to time normal urine is evacuated as a result of occlusion of the ureter upon the diseased side; and by the results of external examination with regard to tenderness, or the discovery of a tumor due to hydronephrosis. In all difficult cases a special examination with the assistance of the newer methods of examination (cystoscopy, separate examination of the urine secreted by each kidney, etc.) is necessary. Admirable diagnostic results are also obtained in cases of renal calculi by examination with the Röntgen rays. With the aid of the new and improved methods of technic, not only phosphatic calculi, but also uratic calculi, can be determined in this manner. In all suspicious cases



RADIOGRAM OF A STONE IN THE RIGHT KIDNEY. (Patient in the dorsal position, the tube being in front and the photographic plate behind him.) *a*, shadow of the twelfth rib; *b*, the renal stone (later removed by operation); *c*, shadow of the lumbar vertebræ.

of any severity, Röntgen photography is therefore urgently to be recommended.

Treatment.—Since the uric-acid concretions are by far the commonest, the methods of treatment most in use for nephrolithiasis refer especially to these.

If the tendency to the formation of urinary gravel be confirmed in a patient, or if the severer symptoms of nephrolithiasis have already appeared, we must first give a number of general dietetic directions, in order to check the formation of uric acid in general, and promote the solution of the uric acid already formed as far as possible. Without entering too much on theoretical reasoning, we will give in what follows the measures which have won general approval. In the first place, the patient must avoid immoderate indulgence in any sort of food, and particularly in meat. Articles of diet, such as liver, sweetbread, and calf's kidneys, which contain an abundance of nuclein, should be forbidden. The patient should be advised to choose a chiefly vegetable diet, with milk and a moderate amount of meat; spirituous beverages are to be used little, if at all, and acid viands and liquids are inadmissible. It is advantageous to weigh the patient regularly in order to keep watch on the nutrition, and to avoid any further increase of weight in such individuals as are well nourished; and in the obese, to bring about a diminution of weight. Moreover, so far as the general condition of the patient allows, he should have regular exercise in the gymnasium, or at sawing wood, or gardening. He should also promote the assimilation of his food by the frequent use of warm baths or salt baths; and an abundance of liquid must be ingested, so as to dilute the urine, and thus increase its solvent power.

This last indication is usually met, in conjunction with that of diminishing the acid reaction of the urine, by the ingestion of alkalies, so as to prevent, as far as possible, the deposit of uric acid; consequently, alkalies and alkaline mineral waters are very extensively employed in nephrolithiasis. Pfeiffer and others have been able to demonstrate by direct experiment that the urine secreted after the use of such waters and drugs does possess an increased power to dissolve uric acid. The simplest way is to have the patient use phosphate of soda in a daily dose of 1 to 4 drachms (gm. 5 to 15); or, better, carbonate of soda, 1 to 2.5 drachms (gm. 5 to 10); or, finally, the specially recommended carbonate of lithia, 2 to 8 gr. (gm. 0.1 to 0.5), several times a day, dissolved in a large amount of simple water, or carbonated water, or lemonade. Also mixtures of these remedies are often used with good results; thus Cantani recommends bicarbonate of soda, 8 gr. (gm. 0.5); effervescing carbonate of lithia, 4 gr. (gm. 0.25); citrate of potassium, 15 gr. (gm. 1); also the so-called uricedin, which is composed of the citrates of sodium and lithium, sulphate of sodium, and chlorid of sodium. Of this, 15 gr. are to be given several times a day. Another remedy is borocitrate of magnesium, of which a teaspoonful may be taken in soda water with sirup, three times a day. Of the natural mineral waters, the springs of Fachingen, Vichy, Carlsbad, Wildungen, Brückenau, Salzbrunn, and Neuenahr are chiefly used. The natural lithia waters of Assmanshausen and Salzschlirf contain such minute amounts of lithia salts that, in general, the artificial waters are to be preferred to them.

The attempt has been often made to promote the solution of uric acid by administering certain chemical substances, but unfortunately the results have not been brilliant. The conditions which obtain in the human organism are

essentially different from those in a test-tube. Piperazin, 15 to 45 gr. (gm. 1 to 3) daily, has been much recommended, but has not proved at all efficient, and the same may be said of lysidin, 15 to 60 gr. (gm. 1 to 4) a day, and urotropin, 15 to 25 gr. (gm. 1 to 1.5) daily, in water. Still, further trial of these remedies, above all urotropin, would not be unjustifiable.

The symptomatic treatment is very important. In so far as this relates to the accompanying pelvic and vesical catarrh, we may refer to the appropriate chapters in this book. For renal hemorrhages some internal remedies, such as ergotin, hydrastis canadensis, etc., have been recommended, but their action is quite doubtful. Subcutaneous injections of adrenalin (one syringeful of a 0.1 per cent solution) deserve somewhat greater confidence. The effectiveness of gelatin injections (℥iij to v [gm. 100 to 150] of a sterilized one- or two-per-cent solution) recommended for various internal hemorrhages, is extremely doubtful. The internal administration of a decoction of gelatin (about five per cent) is more convenient and, therefore, preferable. The treatment of the attacks of colic is of great practical significance. The chief remedies are the narcotics, opium, and morphin, internally, or with very severe pains, better subcutaneously. In severe cases chloral and inhalations of chloroform may be employed. Warm baths, warm poultices, or narcotic embrocations, such as chloroform liniment, also frequently give relief. Sometimes patients experience an alleviation of their pains if the pelvis is raised and the trunk placed in a low position. Some physicians recommend vibratory massage in the region of the kidneys, and cautious downward massage along the course of the ureter, but this does not always agree with the patient. The administration of glycerin (a tablespoonful in tea or milk two or three times), which is claimed to stimulate the contractions of the ureter, must be mainly regarded as a placebo. An abundant supply of fluid is always advisable, in order to aid the washing out of the incarcerated stone by an increased secretion of urine.

Since the initiative of Simon in 1871 the operative treatment of renal disease has made continuous progress, and it has been eminently successful in many cases of renal calculi. It is, therefore, most important in all instances of nephrolithiasis with severe symptoms, and particularly with secondary purulent inflammation, to consider the possibility of surgical interference by nephrotomy or nephrectomy. In the dangerous cases of nephrolithiasis, in which persistent and almost complete anuria occurs, timely surgical interference is of the greatest importance. Particulars in these matters must be sought in special treatises on renal surgery.

What has been said thus far refers, as we have stated, chiefly to the treatment of uric-acid calculi. If there are calculi made up of oxalate, the amount of vegetables eaten must be limited, but the use of alkaline waters is advisable. We do not know any special directions to give in case there were cystin calculi. On the other hand, when phosphatic stones are present, inasmuch as these cannot be deposited unless the urine is alkaline, we should recommend the employment of acids, particularly lactic acid, 8 to 15 gr. (gm. 0.5 to 1), and salicylic acid; also, hydrochloric acid and phosphoric acid. Of course, in most cases there is some disease of the urinary passages which has occasioned the formation of the calculi, and if such disease is discovered its treatment is of paramount importance.

CHAPTER III

TUBERCULOSIS OF THE GENITO-URINARY APPARATUS

Ætiology and Pathological Anatomy.—It does not seem remarkable that, with the presence of many tubercular processes in the body, tubercle bacilli should quite easily reach the kidneys by way of the blood current, and there give rise to an eruption of tubercle. Accordingly, we quite frequently find a few or many miliary tubercles in the kidneys in acute miliary tuberculosis, in pulmonary tuberculosis, etc., which are distributed over the whole kidney, or sometimes only in the territory of one arterial branch.

While miliary tuberculosis of the kidney, however, is without any clinical significance, there is also an extensive local tuberculosis of the kidney, as well as of the urinary tract and the sexual organs. Such affections sometimes occur as a result of pronounced preëxisting tuberculosis of other organs, especially the lungs, or they arise as an apparently independent disease, which is termed genito-urinary tuberculosis. In such cases the infection with the tubercle bacilli often seems to take place by means of the blood from some previously existing—perhaps concealed—tuberculous focus in the body, such as a gland, tuberculous bone or joint disease, etc. In a few cases, the tubercle bacilli perhaps enter the urinary or the genital tract from without. However, a hematogenous infection seems to be the rule in genito-urinary tuberculosis, especially as the results of more recent surgical experience have shown that tuberculous foci generally appear in the kidneys first, and, in fact, almost regularly in only one kidney. The bladder becomes affected only later (exceptionally, in the early stages); the prostate, apparently with particular frequency, and the seminal vesicles and the testicles as well. From the organ first involved, the process then extends continuously or by leaps to the neighboring parts. If the cases come to autopsy, the tuberculosis is often so extensive that we can no longer make out with certainty the place where it first began. In women, tuberculosis is at times predominantly localized in the genital apparatus (uterine and ovarian tuberculosis).

As in all tuberculous affections, individual predisposition plays a part in genito-urinary tuberculosis, which should not be overlooked. This predisposition may be congenital (hereditary or family tendency) or acquired. In regard to the latter, it is to be observed that previous gonorrheal affections (gonorrheal epididymitis, gonorrheal cystopyelitis) and in women, occasionally puerperal pyelitis, prepare the ground for tuberculous infection. In the kidneys the tuberculous infiltration develops either chiefly from the pelvis of the kidney or in the renal substance itself. Yellow cheesy nodules arise, which finally break down and thus lead to an actual "nephrophthisis." If the disease arise from the pelvis, the infiltrated renal papillæ are usually first affected. Ulcerative recesses are formed in the pelvis of the kidney, and finally the entire lining of the pelvis is transformed into a raw surface covered with necrotic tissue and cheesy detritus. In very advanced cases almost the whole kidney is destroyed. As already mentioned, the process is at first one-sided, afterwards usually bilateral, but it is often more advanced on one side than on the other.

If the process invade the ureters, their walls also are infiltrated with tubercular deposits, and hence they are thickened, while the mucous membrane is often changed in great part to a necrotic ulcerating surface. Precisely analogous conditions are found in the bladder, and in some cases even in the urethra; while in the prostate, the vesiculæ seminales, and the testicles there is more frequently the formation of cheesy tubercular nodules, and rarely disintegration and perforation of the tubercular formations.

Clinical Symptoms.—The picture of genito-urinary tuberculosis corresponds in most of its details completely to that of a severe chronic pyelocystitis. The occasional local symptom is pain in the region of the kidneys and bladder. This may sometimes assume great severity, like colic, if the ureter become plugged by a broken-down, crumbling mass. If the bladder is also affected, frequent pressure and pain in urinating arise. In other cases, however, the pain is but slight during the whole disease.

The urine shows the most important diagnostic changes. It almost invariably contains an abundant sediment, consisting of pus corpuscles and detritus. Its amount usually remains normal for a long time; its reaction is faintly acid, but in severe cases it may become alkaline through complication with an alkaline fermentation of the urine. The discovery of shreds of tissue in the urine, elastic fibers and connective tissue, is sometimes possible, and is of diagnostic value because it is direct evidence of an ulcerative process. The discovery of tubercle bacilli in the purulent urinary sediment (Rosenstein and others) is, however, far more important. This is possible in almost all cases, and is a reliable and absolutely decisive factor in diagnosis. There is, however, one unfortunate circumstance about the demonstration of tubercle bacilli in the urine, for not infrequently other bacilli (smegma bacilli) are stained by the ordinary method of staining, which is that employed for sputum, and are thus mistaken for tubercle bacilli. As yet, no easy and certain method of distinguishing these two varieties of bacilli has been discovered, and hence in doubtful cases it is necessary to resort to pure cultures and to inoculation, in order to reach a decision. Still, if the urine is taken by the catheter, and, at the same time, all other phenomena considered, we may, as a rule, manage with the ordinary staining method. [Stain with carbol-fuchsin; decolorize with twenty-per-cent nitric acid; wash in water; and still further decolorize in seventy-per-cent alcohol for at least ten minutes. This will bleach the smegma bacilli.] Other bacilli are usually entirely absent in tuberculous pyuria. Admixtures of blood in the urine are also seen in genito-urinary tuberculosis, but they may often be entirely absent. In several of our cases a slight hæmaturia was the first symptom which called the patient's attention to the trouble with the bladder. Pyuria, however, ordinarily precedes hæmaturia. The more profuse renal hemorrhages generally occur only in the earlier stages of the disease. On the other hand, the microscope will frequently demonstrate a slight admixture of blood. In some cases the urine is entirely free from blood.

The local objective examination of the kidneys usually gives a negative result. Only in a few cases have we been able to feel the diseased kidney as a tumor through the abdominal walls. This is usually due less to the tuberculous infiltration of the kidney itself than to the dilatation of the pelvis of the kidney from hydronephrosis. We can sometimes feel the thickened walls

of the bladder. The local examination of the prostate and the testicles is far more important in diagnosis. Especially in the latter we often feel the hardening corresponding to the tuberculous infiltration, and manifesting itself chiefly in the epididymis, while the hardening and enlargement of the prostate and seminal vesicles can usually be easily detected by rectal palpation.

Among the general symptoms we must mention, first of all, fever, which is only exceptionally absent, and usually, in the severe cases, shows a pronounced remitting, hectic character. The other general symptoms are the same as in most of the other tubercular diseases—anæmia, persistently rapid pulse, emaciation, loss of appetite, increasing bodily weakness, etc. We must be alert to detect the occasional coexistence of other tubercular diseases in the body, the lungs, the intestines, the bones, etc., but these may also be wholly absent, so that we may have to do with a purely local genito-urinary tuberculosis.

The course of the disease is steadily progressive. Recovery does not occur, at least not in any cases where the disease has attained any extent. The disease lasts from a few months to a year or two, but sometimes much longer. The fatal termination usually ensues from the increasing general weakness, more rarely under the symptoms of ammoniæmia, or sometimes from a miliary tuberculosis or some other tuberculous disease, such as pulmonary tuberculosis, tuberculous meningitis, etc., or general amyloidal affection.

Diagnosis.—The diagnosis of genito-urinary tuberculosis is now seldom difficult in fully developed cases, since it can be made with complete certainty by the discovery of the tubercle bacilli joined to the presence of pus in the urine. Of course this gives no information as to the exact local distribution of the process. In order to judge of this, we must add the local symptoms and the physical examination of the different organs. Cystoscopy and catheterization of the ureters have become of great diagnostic importance. Further details will be found in the special monographs. We are aided in the confirmation of our first suspicion of a tuberculous disease chiefly by the consideration of the general condition and the habit of the patient; the discovery of an hereditary taint, or at least the probability of tuberculous infection; and also the discovery of other tuberculous affections, especially the examination of the bladder, the testicles, the prostate and the seminal vesicles, the hectic fever, and the tedious course, upon which nothing has a favorable influence. At any rate we must make it a rule, in every case of apparently spontaneous hæmaturia and especially of persistent pyuria which cannot be otherwise explained, to examine the purulent urinary sediment for tubercle bacilli (*vide supra*). We may then often be able to recognize with certainty the milder and incipient cases of this not very rare affection. [Another valuable diagnostic method, briefly referred to by the author, is by inoculation of 8 to 15 minims of the urinary sediment into the peritoneal cavity of a guinea pig. If tubercle bacilli are present, the animal will develop tuberculosis in six to eight weeks.]

Treatment.—Whether specific treatment of genito-urinary tuberculosis with Koch's tuberculin is likely to have permanent success cannot yet be determined, as so few trials have been made. Nevertheless, it would be justifiable to make a cautious trial of the remedy, although we can hardly cherish the hope that it would prove very beneficial. Beyond this we must resort, in the treatment of tuberculosis of the urinary passages, to the same remedies as in

ordinary chronic pyelitis and cystitis. Of internal remedies we have most frequently used chlorate of potassium, turpentine, creosote, and guaiacol preparations, and have sometimes seen good results, especially from the latter. In vesical tuberculosis it is well to wash out the bladder. Surgical treatment of renal tuberculosis has become of greater importance than the internal. If, by means of an exact examination it can be shown that only one side is affected, the extirpation of the diseased kidney is advisable, and has frequently been accomplished with beneficial results and even permanent cure. Therefore, an early diagnosis is of the greatest importance. Tuberculosis of the testicle is also frequently the subject of surgical treatment.

CHAPTER IV

HYDRONEPHROSIS

(Dilatation of the Pelvis of the Kidney)

Ætiology.—If a contraction arises in any part of the urinary tract and checks the flow of urine, there is a stasis of the urine in the portion behind the stenosis which gradually leads to a constantly increasing dilatation of the tract as a result of the pressure of the retained fluid.

In general, it is evident that gradual constrictions of the urinary tract and periodic obstructions, as from calculi, interrupted by free intervals, lead to more marked degrees of hydronephrosis than rapid and complete obstructions. Under the first-named circumstances the renal secretion persists much longer and is more abundant than in the latter case, when it usually soon ceases. Nevertheless, there may be even then a very slow distention of the pelvis of the kidney, particularly if its mucous membrane continues to secrete. If the obstruction is located in the ureter, the proximal portion of that canal dilates, and still more the pelvis of the kidney, giving rise to what is called hydronephrosis; but if the obstruction is located in the urethra there is a gradual dilatation of the bladder and both ureters, and finally there develops a bilateral hydronephrosis.

A closure of the ureter arises most frequently in adults from impacted renal calculi, and also from new growths in the vicinity, in the uterus or ovaries, which compress the ureter from without. So great a pressure may also be exerted on the ureters by the gravid uterus as to be followed by a hydronephrosis, which is usually bilateral. Cicatricial strictures, valve-formations and bends, also are found in the ureter, and form an obstacle to the flow of urine. Finally, in cancer of the bladder the lower opening of the ureter may be contracted or entirely closed. Constrictions of the urethra, which finally lead to a bilateral hydronephrosis, arise most frequently from strictures as a result of gonorrhea, and also from enlargements of the prostate. In rare cases phimosis may form the obstacle.

It is worthy of note that hydronephrosis may also be congenital, and then it is usually due to congenital defects of development in the ureters or other urinary passages. In later life hydronephrosis is in general more frequently observed in women than in men.

Pathological Anatomy.—The pathological anatomy of hydronephrosis is, on the whole, very simple. We have a dilatation of the pelvis of the kidney, which is associated with a pressure atrophy of the renal tissue. The papillæ are flattened, the uriniferous tubules and the glomeruli are gradually more and more obliterated, and finally the whole kidney may be changed to a connective-tissue sac filled with fluid, and displaying in its walls a few vestiges of the renal parenchyma. The size of such a hydronephrotic sac may sometimes be sufficient to contain 10 or 15 quarts (liters) of fluid. The latter consists, of course, at first of urine, but the farther the atrophy of the kidney advances, the more it merely represents the secretion of the mucous membrane. Inflammatory conditions are found in hydronephrosis only when they have existed previously, as in pyelitis calculosa, or when excitants of inflammation in addition have reached the pelvis of the kidney. We then have a pyonephrosis.

Clinical Symptoms.—Since the whole type of the disease is, of course, dependent in many respects upon the nature of the primary disease, we have here to describe only those symptoms which point to the development of hydronephrosis. Such a condition often causes no special clinical symptoms at all, so that we can at most suspect its existence from the presence of an ætiological factor.

The appearance of a visible and palpable tumor is the first definite point in the diagnosis of hydronephrosis. This first shows itself in the region of the affected kidney, but then it gradually enlarges toward the hypochondrium and the median line of the body, and it may finally reach very considerable dimensions. Hydronephrosis on the left side does not usually move with respiration. On the right side, however, there may be a distinct downward motion upon deep inspiration. The resistance of the hydronephrotic tumor is usually quite considerable, but there may sometimes be a distinct sense of fluctuation. On percussion, the tumor gives a dull note, from which the tympanitic note of the colon in front of the tumor is sometimes distinct (see page 794). It is an important diagnostic sign if the tumor show variations in its size at times—decreasing in size with a simultaneous increase in diuresis, and increasing again when the amount of urine becomes smaller ("intermittent hydronephrosis"). In such cases there is usually a very peculiar alternation in the other clinical symptoms. At the time of the scanty secretion of urine the patient suffers from violent pain, vomiting, chills, and similar symptoms, while upon the appearance of an abundant urinary secretion all these symptoms quickly vanish. In doubtful cases an exploratory puncture of the tumor may also be of significance in diagnosis. It of course favors the assumption of hydronephrosis if urinary constituents, especially urea, can be found in the fluid evacuated; but if the hydronephrosis be of long standing, its contents, as we have said, will be simply seromucous, and then chemical examination gives no definite data for distinguishing hydronephrosis from ovarian tumors or other cystic tumors of the kidney.

The secretion of urine in unilateral hydronephrosis may be completely normal if the other, healthy, kidney acts vicariously. In stricture of the urethra, and also in bilateral constrictions of the ureters, however, there is, of course, an obstacle to the passage of urine, so that the amount of urine may be abnormally small. There may be at times complete anuria, and even

uræmic symptoms. The composition of the urine depends entirely upon the form of the primary disease. If the healthy kidney alone secretes, the urine passed is normal. If there be at the same time pyelitis or cystitis, the urine contains pus or blood. If the urine can also come from the diseased kidney at one time and not at another, the urine, as we have said before, also exhibits a varying composition.

In many cases of hydronephrosis quite severe local symptoms are constantly present; there are frequently severe pains in the tumor, which shoot chiefly toward the thigh. Still, these local symptoms are sometimes surprisingly slight. In regard to the symptoms on the part of other organs, gastric disturbances appear to be of the most frequent occurrence; among them are nausea, loss of appetite, vomiting, and eructations. In some cases the bowels are constipated, in others there is obstinate diarrhea. It is a very interesting fact that, particularly in case of bilateral hydronephrosis, there may be developed a distinct hypertrophy of the left ventricle, with all its sequelæ. The explanation of its occurrence, in our opinion, is precisely the same in this case as in chronic nephritis (*vide supra,* page 741)—viz., the toxic effect of the urinary constituents retained in the blood.

The whole course of the disease is always chronic. There are often variations in its course, but no general statements can be given, because the conditions vary in the different cases according to the form of the primary disease. Most cases of hydronephrosis end fatally, either in consequence of the primary disease or in consequence of secondary pyelonephritic or perinephritic inflammations, of uræmia, etc. Recovery takes place in rare cases, especially if one kidney be perfectly normal, and there be no incurable primary disease. Recovery may ensue spontaneously from perforation or obliteration, or it may be brought about artificially from operative procedures.

Diagnosis.—In the diagnosis of hydronephrosis, the points especially to be considered have already been mentioned. The diagnosis is usually not easy, especially if the ætiological factors be unknown; and the disease is often confused with other renal tumors and echinococci of the kidneys, with ovarian tumors, and even with splenic and hepatic tumors.

Treatment.—Except for the symptomatic treatment of the pain and any accompanying pyelocystitis, an efficient treatment of hydronephrosis can be attempted only by surgical means, the details of which are to be found in the special surgical treatises.

CHAPTER V

CYSTITIS

(Vesical Catarrh)

Ætiology.—In most cases of vesical catarrh the agents of inflammation reach the bladder from without through the urethra. The most unequivocal demonstration in this regard is, unfortunately, often made by the physician himself, when he excites a cystitis by the use of an insufficiently purified and disinfected catheter or bougie. The development of the vesical catarrh is generally aided in such cases by the fact that there is usually a defective

evacuation of urine, from stricture of the urethra or paralysis of the detrusor, with a consequent retention of urine, in which the bacteria can develop undisturbed. The agents of inflammation may also enter from the urethra into the bladder in incontinence of urine. On account of the imperfect closure of the sphincter, a stagnating column of urine, directly connected with the contents of the bladder, forms in the urethra, and to this column the air and the bacteria that excite decomposition of the urine have direct access. In this way many cases of cystitis arise in patients with nervous disease who have paralysis of the bladder, and also in persons who are severely ill and stupid from some other disease, such as typhoid fever.

Cystitis often follows diseases of the neighboring portions of the urinary tract. Among these, gonorrheal urethritis is the most common, and this invades the bladder directly and leads to a gonorrheal cystitis. In this case, is has not yet been absolutely determined whether the gonococci themselves invade the bladder and occasion cystitis, or whether the inflammation is due to the secondary introduction and dissemination of other pyogenic cocci, such as staphylococci (*vide infra*). It is, moreover, a fact of great importance that the development of cystitis is decidedly promoted by the unskillful employment of urethral injections. In women, the agents of inflammation may quite easily enter the bladder from the vagina through the short female urethra. Thus arise especially the frequent cases of cystitis in childbed. In some cases communication may develop between the bladder and certain neighboring organs, as in vesicorectal or vesicovaginal fistulæ, by which again access to the bladder is opened to the agents of inflammation.

Another group of cases is due to the presence of foreign bodies, which irritate the vesical mucous membrane mechanically. Among these is, first of all, the cystitis which so often accompanies stone in the bladder. It must be stated, however, that probably these cases of vesical catarrh are not directly dependent upon the calculi, but are first excited by examination with catheters and sounds, and in brief are due to secondary infection.

In distinction from the methods of origin of cystitis so far described, the production of inflammation by way of the blood supply is much rarer. Certain chemical substances, already mentioned (page 806) which are eliminated by the kidneys and provoke an inflammation of the urinary tract, are the most important in this respect. Cantharides shows the most intense action of this sort, and it may cause an actual croupous cystitis. Slight irritative states of the bladder also frequently appear after taking certain foods and drinks, as after drinking new beer. In the infectious diseases many possibilities have to be taken into consideration. As many bacilli (e. g., typhoid bacilli) are passed with the urine, the possibility of a hematogenous origin of a secondary cystitis is present. On the other hand, the severe illness often occasions an infection by way of the urethra (*vide infra*). It cannot be doubted that in some cases an apparently idiopathic primary cystitis appears after exposure to cold, but it is very rare. In such cases, naturally, the exposure to cold is to be regarded merely as the predisposing cause of the infection or auto-intoxication. Often such cases are but acute exacerbations of an old chronic cystitis—for example, of gonorrheal origin.

As to the special variety of inflammatory germs in cystitis, our knowledge is as yet incomplete. In many cases, particularly in the cystitis of puerperal

women, the germs are perhaps the ordinary pyogenic cocci (streptococcus and staphylococcus pyogenes). In gonorrheal cystitis the invasion of the gonococcus into the bladder has been absolutely demonstrated in some cases. Moreover, the bacterium coli commune seems to play a great rôle in cystitis. In some apparently primary cases of cystitis (as, for example, in children) we must consider the possibility of an infection with the colon bacillus proceeding from the intestine. Moreover, we would state that occasionally colon bacilli appear abundantly in the urine without simultaneous indications of a catarrh of the mucous membrane (pyuria). Such, frequently very obstinate, conditions, termed bacteriuria, have been observed particularly in children. The development of ammoniacal fermentation of the urine (*vide infra*) is, nevertheless, not due to the bacterium coli, but in most instances to special microörganisms. We already know a whole series of organisms which decompose urea (micrococcus ureæ, bacillus ureæ, micrococcus ureæ liquefaciens). In some cases the proteus type is also present.

All these agents of ammoniacal fermentation of the urine probably always get into the bladder from without. They are, therefore, not excitants of inflammation of mucous membranes, although the ammonia formed through their influence certainly does serious damage to the mucous membrane of the bladder.

It has been stated in the previous chapters how frequently cystitis is only one symptom of a more extensive disease of the urinary tract. As cystitis may invade the pelvis of the kidney through the ureters, so, on the other hand, any pyelitis of primary origin may extend downward and involve the bladder.

Pathological Anatomy.—The pathological anatomy of cystitis presents the same conditions as the inflammation of any other mucous membrane. In simple catarrhal cystitis the mucous membrane is swollen and covered with pus, and is often studded with hemorrhages. In chronic cystitis the mucous membrane sometimes takes on a granulated appearance, because of the swelling of numerous follicles, and in other cases assumes a slaty, grayish-black color, because of the pigmentation resulting from numerous small hemorrhages.

The severer forms of cystitis, such as are often observed in diseases of the spinal cord, are termed vesical diphtheria. These cases come to a necrotic destruction of the superficial layers of the mucous membrane, ulcerations, etc. In such severe cases submucous and pericystitic abscesses sometimes develop, which may perforate into the surrounding parts in various ways. The incrustation of the mucous membrane with urinary salts, especially with ammonio-magnesic phosphate, is also frequently found in chronic cystitis, and is worthy of mention. If the disease of the bladder is associated with a stricture of the urethra, the bladder is usually much dilated, and the muscular layer is hypertrophied and stands out in ridges on the inner surface of the bladder.

Clinical Symptoms.—The local symptoms are sometimes quite severe in cystitis, but in other cases they are only slight. In general, they show a greater intensity in acute cases than in chronic cystitis. The pain in the region of the bladder is rarely continuous; it usually occurs only on micturition, but it is often very distressing then, and shoots to the opening of the

urethra. Since the inflamed vesical mucous membrane shows an increased irritability, and since the morbidly altered urine (*vide infra*) also exerts an abnormal irritation on the mucous membrane, there is very often an increased desire to micturate. The patient has to empty the bladder much oftener than normal, and in severe cases there is an almost constant, painful "vesical tenesmus," and at every attempt to micturate only a very small amount of urine is passed, with severe burning. As a result of the increased irritability of the vesical mucous membrane, there sometimes comes on a very troublesome reflex spasm of the sphincter, by which the symptoms are increased.

The character of the urine is alone decisive in the diagnosis. This is secreted in a perfectly normal amount and quality, in case there is no complication on the part of the kidneys; but in the bladder it is mixed with the products of the diseased mucous membrane, and it is here exposed to the action of the bacteria in a way that will presently be described. The abnormal constituents of the urine are chiefly pus corpuscles, bladder epithelium, and mucus secreted by the mucous membrane. As a rule, the urine of cystitis contains innumerable bacteria, under the influence of which (*vide supra*) develops that important chemical transformation of urea into carbonate of ammonia, which is characteristic of all severe cases of cystitis, and which has been termed the alkaline (ammoniacal) fermentation. This formation of carbonate of ammonia decidedly aggravates the inflammation. As Lépine and Roux have shown, we can produce a severe cystitis, and even nephritis, by injecting very small amounts of a pure culture of micrococcus ureæ into the bladder of a guinea pig. The stagnation is a factor which greatly aids the whole process, since the activity of the bacteria, as we have said, can develop much better than if the bladder were to a certain degree constantly purified and washed out by the urine; but cystitis cannot be produced by mere retention of urine.

Fig. 102.—Crystals of triple phosphate and ammonium urate. (From Funke.)

As soon as a part of the urea is changed to carbonate of ammonia, the urine must be less acid in reaction. The urine has a faintly acid or neutral reaction, and sometimes it is decidedly alkaline when passed. The latter, however, is only rarely the case, but it is often simulated by the fact that the urine is not examined until it has stood for some time. Since during this time the alkaline fermentation makes rapid progress, the urine that has stood is very often alkaline. Many crystals of ammonio-magnesic phosphate and urate of ammonium then form in it; the former are easily recognized by their "coffin-lid shape," and the latter by their "thorn-apple shape" (see Fig. 102). There is also a deposit of amorphous phosphates.

If we then briefly sum up what has been said, the urine is passed in about the normal amount in cystitis. It usually looks yellow, and has an abundant sediment, which can often be recognized as purulent with the naked eye,

and in which, microscopically, we can find pus corpuscles, often bladder epithelium, and constantly innumerable bacteria—usually short rods in vigorous motion. The alkaline fermentation may usually be recognized by the strong ammoniacal odor, and also, as we have said, by the reaction of the urine. In the severe diphtheritic forms of cystitis we find shreds of necrotic tissue in the urine. If there are hemorrhages in the bladder, the urine often contains red blood corpuscles and sometimes even large blood clots. The mucus in the urine appears in milder cases as a cloudy opacity—"*nubecula.*" The viscid masses which can be drawn out into threads, and which are usually abundant in the urine in severe cystitis, and sometimes form gelatinous casts corresponding in shape to the bottom of the vessel containing the urine, are not mucin, but they arise from the pus corpuscles and the epithelium, changed and dissolved in the alkaline urine, and hence give the reactions for albumen. It goes without saying (compare page 727) that cystitic urine is always albuminous from its mixture with pus serum. The presence of slimy threads in the urine—the so-called "clap-threads" (*Tripperfäden*)—is characteristic of gonorrheal cystitis.

There can be no doubt that, as already stated, the decomposing alkaline urine acts as a chemical irritant on the vesical mucous membrane. Hence cystitis often arises perhaps in this way, that the bacteria which have entered the bladder first excite only an alkaline fermentation, and that then the mucous membrane is affected by the irritation of the ammonia salts that are formed. Sometimes, however, the mucous membrane endures this irritation for a long time, and this explains the fact that alkaline fermentation may be observed in cases which, upon autopsy, present an almost normal condition of the vesical mucous membrane. In addition to the alkaline fermentation of the urine, it seems probable that many other processes of disorganization may develop in the urine, as is indicated by the various sorts of bad odors which the urine of severe cystitis may have. In a few cases there is even a formation of gas (carbonic dioxid, nitrogen, hydrogen), giving rise to pneumaturia. This phenomenon indicates the development of special bacteria which generate gas. If the patient is also suffering from diabetes mellitus, the pneumaturia may be due to the fermentation of the sugar in the bladder.

The other morbid symptoms associated with cystitis usually depend only in part upon the disease itself and in part upon some existing primary disease. The most important symptom is the fever, which is often to be referred directly to the cystitis. In severe cases it may be very intense, and often assumes a pyæmic intermittent character, especially if there have arisen pericystitic suppurations or if the cystitis has extended to the pelvis and parenchyma of the kidney (see page 788). An acute cystitis also may begin with a chill and high fever. If the escape of the purulent urine, however, is absolutely free, fever may be entirely absent in spite of the existence of cystitis.

Sometimes in severe cystitis, with a marked alkaline fermentation, certain nervous symptoms appear, such as headache, vertigo, stupor, and nausea. The idea has been advanced that in these cases we have to do with an auto-intoxication of the body, since ammonia and perhaps other products of decomposition, such as sulphureted hydrogen (?), are absorbed from the bladder into the blood (ammoniæmia), and in this way excite the symptoms of poisoning mentioned.

According to the course of the disease we distinguish an acute and a chronic cystitis. The former, which may come on, for example, after catheterization, in gonorrhea, etc., often terminates favorably after a few days or weeks. The amount of mucus and pus in the urine is never great. Chronic cystitis is observed especially as a complication in other diseases of the urinary tract, such as stricture, chronic diseases of the spinal cord with paralysis of the bladder, etc. It is very often incurable because the primary disease is incapable of improvement and the cause of the disease therefore persists. The longer a cystitis lasts, the greater is the possibility of the development of severe and dangerous complications, especially the development of a pyelonephritis, and the formation of pericystitic suppurations. In this way cystitis, especially in chronic nervous diseases, may become the immediate cause of death.

Treatment.—The dangers last mentioned must urgently impress upon us the prophylaxis of cystitis. Fortunately, a good deal can be done in this respect, in the first place, by the avoidance of all unnecessary use of bougies and catheters, by the greatest cleanliness in the use of all instruments of this sort, and by the timely treatment of all those conditions which may lead to cystitis.

The treatment of cystitis is, in the milder and acute cases, hygienic and medicinal, but the severer cases demand careful local treatment.

In any severe, and especially in any acute, cystitis, the greatest bodily rest (if possible rest in bed) is urgently desirable, since otherwise an increase of the symptoms and a prolongation of the course of the disease is the almost inevitable result. The diet must be mild and unirritating. Spiced food and alcoholic drinks are to be avoided, but we should recommend an abundant supply of fluid, by which the urine is diluted and the bladder washed out.

We have the patient drink plenty of ordinary water, tea (made from uva ursi, etc.), or a suitable mineral water, like Wildungen, Fachingen, Wernarzer, etc. A diet mainly of milk is very good; under it, improvement is often surprisingly rapid.

Among internal remedies those are to be considered which are eliminated with the urine, and are thus able to act on the diseased mucous membrane, or directly upon the agents of inflammation and the ammoniacal fermentation. One of the most efficient drugs, which never does harm if proper caution is exercised, is chlorate of potassium, of whose favorable influence on vesical catarrh we have often convinced ourselves. It is prescribed in an aqueous solution, 40 to 75 gr. (gm. 3 to 5) a day, and it should never be taken on an empty stomach. Far less efficient than chlorate of potassium are the astringents, such as tannin, tannigen, tannalbin, and uva ursi. The latter drug is said by Lewin to contain a glucosid, arbutin, which is itself recommended in doses of 45 to 60 gr. (gm. 3 to 3) a day, but its efficacy is doubtful. Of metallic astringents, acetate of lead in powders of 0.5 to 1.5 gr. (gm. 0.03 to 0.1), repeated several times a day, is at present seldom employed. In cases with a decided purulent secretion, if the initial symptoms of irritation have abated, we may often prescribe with good results resinous drugs, of which the best are oil of turpentine, in capsules or in milk, or balsam of copaiba.

All of these, however, have latterly been displaced by urotropin, which

is best prescribed in powders of 7.5 gr. (gm. 0.5) three to four times daily, or even in solution (5 parts in 150), three tablespoonfuls daily. Helmitol, though very similar, is less effective. Urotropin is the best remedy in every bacteriuria. In addition, salicylic preparations, especially salol, in powders of 8 gr. (gm. 0.5), repeated several times a day, are specially to be recommended; naphthalin, 1.5 to 5 gr. (gm. 0.1 to 0.3), repeated several times a day, has also been recommended, but it may produce decidedly disagreeable symptoms.

If there are severe local symptoms, we prescribe warm compresses and poultices to the region of the bladder. In other respects narcotics, especially subcutaneous injections of morphin, are the best remedy when there is severe pain and tenesmus. Morphin, as well as opium and belladonna extract, are also employed to good advantage in the form of suppositories. Finally, the frequent use of protracted warm baths may be greatly recommended.

In chronic cystitis all the remedies previously mentioned are also to be considered; but they are usually not sufficient alone, and at any rate they are far less effective than a methodical local treatment. This consists in a regular daily washing of the bladder by means of an elastic catheter. A rather long rubber tube is attached to the catheter at one end, and to a funnel at the other, and by raising or lowering this funnel the bladder can be filled or emptied. We allow a moderate amount, say 3 to 7 ounces (100 to 200 c.c.), of the warm fluid to run into the bladder and escape, repeating the process until the wash water is clean. For this, perhaps, we employ either a simple one-half to one-per-cent solution of common salt, or preferably a warm and weak solution of acetate of lead, 1 to 1,000; permanganate of potassium, 1 to 1,000; or a combination of boric and salicylic acids. By treatment of this sort many cases of chronic vesical catarrh may be cured, while the most obstinate may, at any rate, be kept within bounds. In rebellious cases of purulent cystitis it is advantageous to employ lunar caustic. The bladder is rinsed out, and then about 5 ounces (gm. 150) of a weak solution of nitrate of silver, at first 1 to 3,000, later as strong as 1 to 1,000 or 1 to 500, are injected through a catheter. The solution is allowed to remain two or three minutes in the bladder and then withdrawn. What is called the Janet method of irrigating the bladder is also very useful. The fluid is placed in a fountain syringe, and made to enter the bladder from the urethra, by raising the reservoir high enough to give a pressure which overcomes the sphincter of the bladder. The advantage of this method lies in the coincident dilatation and lavage of the posterior portion of the urethra. [An excellent remedy is argyrol. Wash out with a solution 1 to 1,000, then leave an ounce of five- to twenty-per-cent solution in the bladder.]

It is very important, in treating chronic catarrh of the bladder, to consider the possible cause of the condition—for instance, stricture, or vesical calculi, or paralysis of the bladder.

In pericystitic suppuration surgical treatment is only rarely possible. We must, therefore, confine ourselves to purely symptomatic procedures.

CHAPTER VI

NEW GROWTHS IN THE BLADDER

1. **Cancer of the Bladder.**—Primary carcinoma of the bladder is rare. It forms localized tumors, sometimes with a pedicle; or it extends diffusely over the wall of the bladder, and in that case leads to such thickening that the bladder can sometimes be felt through the abdominal walls as a firm tumor. Secondary carcinoma of the bladder is not infrequently due to direct involvement by extension from neighboring parts, such as the uterus, prostate, or rectum.

The early symptoms of primary cancer of the bladder consist of disturbances of micturition, which are often difficult to explain. Usually the first indication of the special local disease consists of a change in the character of the urine, which is probably coincident with the beginning of ulceration in the tumor. Then the complete picture of severe chronic cystitis is rapidly developed, a particularly characteristic symptom being the frequent, though irregular, appearance of blood in the urine. The condition becomes most distressing if the vesical termination of the urethra is occluded by the new growth. In the cases which we have seen, cancerous cachexia did not develop until rather late. One case was in the person of a quite young man. In general, the entire course of the disease seldom occupies more than one or two years.

The *diagnosis* of vesical cancer is not always easy, particularly at first. The main point is, that when there is a chronic disease of the bladder and no other sufficient reason for it is discovered, we should think of the possibility of tuberculosis or cancer, and make a careful examination with these severe conditions in mind. It is sometimes possible to confirm the diagnosis by finding bits of tumor in the urine, but this is not invariably possible; consequently a number of methods have been elaborated to facilitate early recognition of vesical tumors by means of a direct and careful examination of the bladder—thus we have: cystoscopy (Nitze); exploratory cystotomy; and in women dilatation of the urethra and digital examination of the bladder.

Particulars with regard to these procedures and their value are to be found in monographs, and in them the reader can also inform himself as to the results of the attempts which have thus far been made to remove vesical tumors by operation.

2. **Papilloma.**—Papilloma of the bladder is usually a very soft fibroma, which is located in the trigonum or fundus and has delicate waving papillæ, which are covered with several layers of cylindrical epithelium. The local discomfort which this not very rare form of tumor occasions is often slight, but the hemorrhage it causes may be very important, being chronic and obstinate; for months and years the hæmaturia may be more or less continuous. The clotted blood as it passes through the urethra often assumes the shape of a worm. There is not much disturbance of micturition unless portions of the tumor obstruct the inner end of the urethra. Anyone who has had the experience which we have had of seeing an otherwise healthy and vigorous man die because of persistent hemorrhage from a papilloma hardly as big as a walnut, cannot insist enough upon the value of an early and certain diag-

nosis (reached by examining portions of the tumor, or, still better, by cystoscopy) and of surgical treatment.

CHAPTER VII

ENURESIS NOCTURNA

(Nocturnal Incontinence of Urine)

Enuresis nocturna is a nervous affection of the bladder by no means rare in children of both sexes, and therefore quite important in its practical relations. Of course, in small children there is no sharp boundary to be drawn between normal and pathological conditions; but it is decidedly pathological if larger children, from four to ten years of age and even older, pass their urine in bed more or less frequently during sleep, in spite of well-developed reasoning powers and professedly the best intentions. This may extend to the years of puberty and even beyond, and then it frequently produces a very depressing mental influence upon the patient. Special causes for it are not to be discovered in most cases. We are compelled to assume either an abnormal weakness of the sphincter, which is probably sometimes congenital, or an abnormal irritability of the detrusor. Most frequently, but not invariably, the child has a neurotic constitution. At any rate, in wetting the bed at night the process of micturition comes on in a purely reflex way, but it is often accompanied by certain ideas in dreams referable to micturition. It does not hold in all cases that the sleep is especially deep. Many patients, of course, do not notice the mishap till morning, but others almost always wake directly after. The involuntary micturition usually occurs in the first hours after going to sleep, but sometimes it is later, and even toward morning. By day micturition is often perfectly normal; but in many cases there is even then a noticeable weakness of the bladder, so that the child has to make water oftener than usual, and sometimes even wets its clothes.

Although, as we have said, we can usually find no special cause for the trouble, still, in some cases, certain morbid changes in the urinary organs may give rise to the incontinence. We should, therefore, in every case at least think of the possibility of stone in the bladder, of congenital phimosis and adhesions of the prepuce to the glans penis, of ascarides, of inflammatory conditions, and foreign bodies in the vagina, and make a special examination into these points. We must also bear in mind polyuria caused by diabetes or renal disease, and finally, of course, in the diagnosis of a purely nervous nocturnal incontinence of urine, we must exclude the existence of any actual anatomical spinal affection.

In all the cases just mentioned, the treatment must be directed first to the primary disease; but in the ordinary nocturnal incontinence the treatment must be aimed at the symptom of nocturnal micturition as far as possible. The child must take only a very little fluid in the evening, and he should be made to empty his bladder immediately before going to sleep, and perhaps once again later. He should not be covered up too warmly, and, if possible, he should not lie on his back during sleep. Tying a brush to the back is there-

fore a well-known domestic remedy. It is strongly recommended that the child should be made to lie in bed with the hips elevated and the head low. A somewhat strict mental treatment is often effective, since thus the attention to the process is increased, although unconsciously, and the child often learns to wake up at the right time. We have seen many cases of enuresis, which did not recover when treated at home, get well quite rapidly when the children were isolated in the hospital. The employment of the rod is rarely allowable; on the other hand, we often must shield the child from injudicious parents.

Internal remedies, such as belladonna and tincture of nux vomica, were formerly recommended, but they seldom do good. Lately the tincture of rhus aromatica has been employed again; of this, 15 drops are given in the afternoon and in the evening before going to bed. In anæmic children preparations of iron are indicated. It is often useful to employ electricity, although it may be that the only influence it has is psychical. We put the broad anode over the lumbar cord, and the smaller cathode over the region of the bladder or on the perineum, and let quite a strong constant current pass through for two or three minutes. The current may also be interrupted and closed a few times. Then we pass the wire end of one conducting cord, which we make the cathode, into the mouth of the urethra for 1 or 2 cm., while we place the other broad electrode on the perineum or above the symphysis, and let quite a strong and somewhat painful faradic current act for one or two minutes (Seeligmüller). The sittings must at first be repeated daily. It is also a very good plan to let the whole body be well rubbed with cold water before going to sleep. It is said that in obstinate cases the introduction of large bougies and the consequent distention of the posterior portion of the urethra are often attended with rapid improvement.

Even in cases in which, despite careful treatment, no permanent success is attained, we have this consolation with regard to the prognosis, that in simple enuresis the abnormal condition usually undergoes gradual and spontaneous improvement as the patient grows older.

[Belladonna, strychnin, or nux vomica, or a combination of the two, are often of unquestionable service. If the enuresis is only nocturnal, belladonna alone may be used, either in a single dose at bedtime or three times a day. If the enuresis is diurnal also, the two drugs should be combined and given three or four times a day.]

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